

DISEASES OF THE HEART

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WITH A CHAPTER ON

THE OCULAR MANIFESTATIONS OF ARTERIAL DISEASE

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THIS EDITION IS DEDICATED
WITH FILIAL PIETY
TO
THE ROYAL INFIRMARIES
OF
EDINBURGH AND GLASGOW

PREFACE TO THE SECOND EDITION

THE present edition is virtually a new book. Many of the chapters are entirely new, while the remainder have been revised in the light of the advancement of our knowledge since 1914. It is, however, in the main still a personal record, based upon our own experience, although we have not failed to make use of the work of other writers.

During the last eight years the action of the heart in health and in disease has been the subject of world-wide study. Old problems arose anew during the war. The significance of cardiac murmurs, the functional efficiency of the heart, the effects of mechanical strain and infective disease were again investigated, and the results revealed considerable diversity of thought and of teaching. A more complete study of the heart's action in disabled soldiers has elucidated some of these problems.

Recent physiological researches have shed fresh light on the work which the heart performs, and on the manner whereby it adapts itself to the varying needs of the body. The mechanism of its nervous control is more clearly understood. The phenomena of dyspnoea and cyanosis have been re-investigated, and also the bacteriology of acute endocarditis. The importance of focal infection in the mouth, throat and elsewhere, as a potential cause of cardiac disease, is more fully appreciated. The Wassermann reaction has shown that the influence of syphilis as a causal factor in disease of the heart and the arteries was formerly underestimated, and the value of anti-syphilitic treatment in appropriate cases has been proved. The effects of digitalis have been still further studied, and its uses have been standardized.

The electrocardiograph has continued to yield valuable information concerning the irregularities of the heart's action,

and the effects of myocardial lesions. The lapse of time has permitted the collection of data with regard to certain cardiac disorders, and their analysis has led to greater precision in diagnosis, prognosis, and treatment.

A change has been made in the arrangement of the text. The opening chapters deal with recent anatomical and physiological work and with the pathology of the myocardium. There follow chapters on the examination of the heart; the disturbances of its rhythm; acute endocarditis, chronic valvular and myocardial disease, angina pectoris, "D.A.H.," pericarditis, congenital disease of the heart, the diseases of the arteries and aneurysm of the aorta. A. J. Ballantyne has again kindly contributed the chapter upon the ocular manifestations of arterial disease.

Many new illustrations have been introduced. With three exceptions (Figs. 63, 259, 269), they are original. The pencil drawings are by A. K. Maxwell; the drawings of microscopic sections are by Richard Muir.

With grateful thanks we acknowledge our indebtedness to many friends: the curators of the Museums of the Royal College of Surgeons of Edinburgh, of the Royal College of Surgeons of England, of the Royal Infirmary of Glasgow and of the Western Infirmary, for their permission to utilize specimens for illustrative purposes; the trustees of the Clinical Medicine Research Laboratory of the Royal Infirmary of Edinburgh for facilities in the study of cases; the Editors of the *Edinburgh Medical Journal* for Figures 63 and 259; Katharine M. Chapman, J. R. Riddell, H. L. Watson Wemyss and J. D. Gunn, for new illustrations; Professor Lovell Gulland, who has granted us every facility to study the patients in his wards; J. G. A. Burton, J. Gibson Cattanach, H. Whitridge Davies, J. W. Dowden, J. K. Rennie; another series of residents; and lastly, as before, Farquhar Macrae.

PREFACE TO THE FIRST EDITION

DURING the last ten years great advances have been made in our knowledge of the diseases of the heart and arteries. New methods of histological technique have revealed lesions which were hitherto unappreciated, and experimental research has deciphered their causes. The sphygmomanometer, the polygraph, the electro-cardiograph, and the Röntgen rays, have become accessible to the clinician, and the data thus acquired have elucidated some of the many problems which awaited solution; while the pharmacologists have defined the uses of such drugs as digitalis more accurately than had been previously possible.

The following pages are an attempt to review the whole subject in the light of these recent advances, and to present to the practitioner the results which have been attained, and their bearing upon the practical work of *diagnosis*, *prognosis*, and *treatment*. They are based in the main upon my personal experience and the records in my own wards, though I have utilized, where it seemed desirable, the numerous monographs and articles which have recently appeared, and the experience of my medical friends.

The importance of the cardiac muscle in the maintenance of the circulation renders necessary a short account of the diseases by which it is affected. Arterial disease and its influence upon the heart are discussed in the three succeeding chapters. The myogenic theory and the various disturbances of cardiac function are considered at some length, and are accompanied by an account of the electro-cardiograph. Acute endocarditis, chronic valvular disease, myocardial weakness, and pericarditis then follow in order.

The chapters so kindly contributed by W. T. Ritchie and

A. J. Ballantyne are of special value, as they are written by recognized authorities upon these particular subjects.

The illustrations are mainly original, and I must express my indebtedness to the kindness of Professors Arthur Keith, Robert Muir, and J. H. Teacher for their permission to utilize the specimens in their museums. The former has also allowed me to reproduce three of his diagrams. The microscopic pictures have been made from my own collections and those of A. M. Kennedy and A. W. Harrington. The skiagrams were taken from my patients by J. R. Riddell.

The polygraph tracings are, with one exception, from my own collection. This I have done purposely, even in cases where more perfect examples might have been obtained elsewhere, in order to show the frequency with which disturbances of cardiac function occur in the ward work of a general hospital, and the possibility of recording them in a busy clinique. Several of them were obtained—unaided—by my residents.

I have not attempted to supply a full bibliography, and the references which are given refer to statements which may not yet be generally accepted or known, or add point to the text. My own papers will be found to contain numerous references to their particular subject. I have made little reference to standard works, but I must acknowledge my indebtedness to the writings of G. W. Balfour, Sir W. Broadbent, A. R. Cushny, G. A. Gibson, W. H. Gaskell, H. Huchard, James Mackenzie, A. E. Sansom, W. Stokes, and Graham Steell; and to the articles in Sir Clifford Allbutt's and in Sir William Osler's "Systems."

I have great pleasure in acknowledging the kind assistance in my task of many friends, in particular Robert Muir, and my collaborators and colleagues, W. T. Ritchie, Archibald W. Harrington, A. M. Kennedy, J. R. C. Greenlees, David MacDonald, and G. B. Fleming; my past and present residents, without whose willing aid my records must of necessity have been scanty; and last, but not least, Farquhar Macrae.

CONTENTS

CHAPTER I

ANATOMY

	PAGE
The myocardium. The coronary circulation. The nodes, the bundle and its branches. Relation of the auriculo-ventricular node and bundle to the membranous septum and to the valvular orifices. Structure of the nodes and bundle	1-16

CHAPTER II

PHYSIOLOGY

The contraction of the heart. Properties of the cardiac muscle.	
The regulation of the action of the heart. The nervous control of the heart. The action of the vagus. The action of the sympathetic. The mechanical control of the heart. The reserve power of the heart. Dyspnœa. Respiratory exchange	17-30

CHAPTER III

THE DISEASES OF THE MYOCARDIUM

Hypertrophy. Atrophy. Ischæmic atrophy. Granular degeneration. Hyaline degeneration. Fatty infiltration. Fatty degeneration. The fibroses of the heart. Infaret. Para-arterial fibrosis. Peri-arterial fibrosis. Mixed forms of fibrosis. Syphilis. Aneurysm of the heart. Rupture of the heart. Dissociation	31-55
---	-------

CHAPTER IV

THE EXAMINATION OF THE HEART

Polygraphic curves: their interpretation. The apex impulse. The arterial tracing. The cervical curve. The electrocardiograph. The electrocardiographic deflexions	56-76
---	-------

CHAPTER V

STIMULUS PRODUCTION

Sinus tachycardia. Sinus bradycardia. Sinus irregularity	77-86
--	-------

CHAPTER VI

EXCITABILITY

	PAGE
Extrasystoles: Frequency. Etiology. Symptomatology. The arterial pulse. Ventricular extrasystoles. Auricular extrasystoles. Nodal (auriculo-ventricular) extrasystoles. Coupled rhythms. Diagnosis of extrasystoles. Prognosis of extrasystoles. Treatment	87-112

CHAPTER VII

CONDUCTIVITY

(1) Auriculo-ventricular conductivity: Depression of conductivity; partial auriculo-ventricular block; complete auriculo-ventricular block. The Adams-Stokes syndrome. Diagnosis. Prognosis. Treatment. (2) Bundle-branch block. (3) Intraventricular (arborization) block. (4) Sino-auricular block	113-141
--	---------

CHAPTER VIII

CONTRACTILITY

Defective contractility. Alternation. Associated symptoms and signs. Diagnosis. Prognosis. Treatment	142-150
--	---------

CHAPTER IX

TONICITY

Arguments for and against tonicity. Depression of tonicity. Symptoms and signs. Dilatation of heart. Treatment	151-155
--	---------

CHAPTER X

AURICULAR FLUTTER

Historical. Etiology. Morbid anatomy. Experimental flutter. Clinical features. Flutter in an apparently healthy heart. Flutter in the course of chronic heart disease. Diagnosis. Prognosis. Treatment. Action of digitalis and strophanthus	156-173
--	---------

CHAPTER XI

AURICULAR FIBRILLATION

Historical. Etiology. Age and sex incidence. Co-existing disease. Nature of fibrillation. Clinical features. Mode of onset. Severity of symptoms. The arterial pulse. The heart. The cardiac sounds. Venous pulsations. Electrocardiograms. Diagnosis. Prognosis. Treatment. Digitalis. Quinidine	174-197
---	---------

CHAPTER XII

NODAL (AURICULO-VENTRICULAR) RHYTHM

	PAGE
Etiology. Clinical features. (1) Nodal rhythm without tachycardia. (2) Continuous nodal tachycardia. (3) Paroxysmal nodal tachycardia. Paroxysmal ventricular tachycardia. Prognosis. Treatment	198-214

CHAPTER XIII

ACUTE ENDOCARDITIS

The lesions. Etiology. (1) Acute simple endocarditis. (2) Malignant endocarditis. Bacteriology	215-229
--	---------

CHAPTER XIV

THE SYMPTOMS OF ACUTE ENDOCARDITIS

(1) Simple endocarditis. The pulse. Cardiac overaction. Auscultatory signs. Subcutaneous nodules. Fever. (2) Malignant endocarditis. Embolism	230-240
---	---------

CHAPTER XV

THE DIAGNOSIS, PROGNOSIS AND TREATMENT OF ACUTE ENDOCARDITIS

Diagnosis. Prognosis. Treatment. Rest. Salicylates. Sera. Vaccines. Prevention of re-infection. Drugs	241-251
---	---------

CHAPTER XVI

CHRONIC VALVULAR DISEASE

The lesions. Stenosis and incompetence of valve. Etiology : (1) Acute endocarditis. (2) Syphilis. (3) Mechanical strain. Associated renal disease. (4) General cardio-vascular degeneration. (5) Congenital defects	252-274
---	---------

CHAPTER XVII

THE SYMPTOMS OF CHRONIC VALVULAR DISEASE

The causes of symptoms. Dyspnoea. Cheyne-Stokes breathing. Paroxysmal dyspnoea. Cough and expectoration. Cardiac discomfort and pain. Palpitation. Oedema. Gastro-intestinal symptoms. Embolic phenomena. Jaundice. Cerebral symptoms. The urine. The symptoms of mitral disease. The symptoms of aortic disease	275-306
--	---------

CHAPTER XVIII

THE DIAGNOSIS OF CHRONIC VALVULAR DISEASE

The significance of physical signs. Pulsation on the front of the chest. Area of cardiac dullness. The causes of murmurs.

	PAGE
Canter rhythm. The physical signs of mitral disease : (1) Mitral stenosis ; (2) Mitral incompetence. The physical signs of aortic disease : (1) Aortic stenosis ; (2) Aortic incompetence. Physical signs of valvular disease of the right heart. Pulmonary stenosis. Pulmonary incompetence. Tricuspid incompetence. Tricuspid stenosis	307-347

CHAPTER XIX

PROGNOSIS OF CHRONIC VALVULAR DISEASE

The nature of the lesion. The site of the lesion. The degree of the lesion. The rhythm of the heart. The presence of disease in other viscera. The degree of compensation. The cause of failure. Age. Sex. Social circumstances . . .	348-357
---	---------

CHAPTER XX

MYOCARDIAL FAILURE WITHOUT VALVULAR DISEASE

Myocardial failure in acute infective diseases. Fatty changes. Disease of the coronary arteries. Myocardial fibrosis. The physical signs of myocardial failure . . .	358-369
--	---------

CHAPTER XXI

THE TREATMENT OF CHRONIC CARDIAC FAILURE

Rest. Diet. The treatment of symptoms. The action of digitalis. The use of digitalis in auricular fibrillation. The use of digitalis in flutter. Strophanthus. Caffeine. Theobromine. Diuretics. Stimulants. Antisyphilitic treatment	370-387
---	---------

CHAPTER XXII

ANGINA PECTORIS

Pain. Its site and severity. The rhythm of the pulse. Etiology. The elevation of blood-pressure. Weakness of the heart. Toxic angina. Pathology. Diagnosis. Prognosis. Treatment	388-399
--	---------

CHAPTER XXIII

DISORDERED ACTION OF THE HEART

Symptoms. Etiology. Interpretation of symptoms . . .	400-407
--	---------

CONTENTS

xv

CHAPTER XXIV

ACUTE PERICARDITIS

	PAGE
The lesions. Etiology. The infections. Intrathoracic disease. Pyæmia. Terminal pericarditis. The symptoms. The effects of effusion. The physical signs. Latent pericarditis. Prognosis	403-427

CHAPTER XXV

ADHERENT PERICARDIUM: PNEUMO-PERICARDIUM

Indurative mediastino-pericarditis. Pericarditis interna et externa. Etiology. The symptoms. The physical signs. Illustrative cases. Pneumo-pericardium. Etiology. Physical signs. Prognosis	428-448
--	---------

CHAPTER XXVI

THE TREATMENT OF PERICARDITIS

The treatment of pericarditis	449-452
---	---------

CHAPTER XXVII

CONGENITAL HEART DISEASE

Development of the heart. Dextrocardia. Defects of the inter-auricular septum. Defects of the inter-ventricular septum. Pulmonary stenosis. Patent ductus arteriosus. Tricuspid, and mitral, stenosis and incompetence. Diagnosis of congenital heart disease. Prognosis. Treatment	453-468
---	---------

CHAPTER XXVIII

THE DISEASES OF THE ARTERIES

The arteries. <i>Arterio-sclerosis</i> . The lesions. Etiology. High blood-pressure. Chronic renal disease. Increased viscosity of the blood. Polycythæmia hypertonica. Plethora. The infections. <i>Focal lesions</i> . Atheroma. Mesarteritis End-arteritis obliterans. Medial calcification. Etiology	469-494
--	---------

CHAPTER XXIX

THE SYMPTOMS OF ARTERIAL DISEASE

High blood-pressure. Cardiac hypertrophy. Consecutive mitral disease. Uræmic symptoms. Emphysema. Dyspnoea. Cerebral symptoms	495-513
---	---------

CHAPTER XXX

THE OCULAR MANIFESTATIONS IN ARTERIAL DISEASE

The retinal vessels. Miliary aneurysms. Retinal hæmorrhages.	PAGE
Albuminuric retinitis. Obstruction of the central artery and of the central vein	514-520

CHAPTER XXXI

THE TREATMENT OF ARTERIO-SCLEROSIS

Preventative treatment. Disturbance of metabolism. Elimination. Diet. Laxatives. Baths. Exercise. Massage. Drugs. Vaso-dilators. Digitalis. Stimulants. Diuretics. Blood-pressure charts. Iodides. Mercury	521-543
--	---------

CHAPTER XXXII

ANEURYSM OF THE AORTA

Etiology. Syphilis. Age. Sex. Occupation. Morbid anatomy. <i>Aneurysm of the arch of aorta</i> : Circulatory symptoms and signs. Radiographic examination. Respiratory symptoms. Nervous symptoms. Alimentary symptoms. Other symptoms. <i>Aneurysm of the descending thoracic aorta</i> . <i>Aneurysm of abdominal aorta</i> . The course of aortic aneurysm and mode of death. Diagnosis. Prognosis. Treatment	544-585
INDEX	587-605

COLOURED PLATES

FIG. 24. Fibrosis of the anterior wall of the left ventricle	TO FACE PAGE 29
FIG. 29. Marked narrowing of the orifices of the coronary arteries from syphilitic disease of the aorta. Infarct, with fatty degeneration of the walls of the left ventricle	43
FIG. 292. Right fundus oculi from a case of chronic nephritis	519

FOLDING PLATES

FIGS. 112-116	129
FIG. 193	281

DISEASES OF THE HEART

CHAPTER I

ANATOMY

The Myocardium.—The cardiac muscle is very specialized and differs in many respects from voluntary and involuntary muscle elsewhere. The cells are irregular in shape, more or less rhomboidal on longitudinal section, and divide into branches which unite directly with those of adjacent cells. There is no sarcolemma, and while appropriate staining shows the junctions of the adjacent cells very distinctly, these are invisible in unstained sections, and many observers are inclined to regard the cardiac muscle as forming a syncytium.¹ The nucleus is large and oval in shape, with a well-marked chromatin network, and lies in the centre of the cell bounded laterally by the fibril bundles. At each pole lies an ovoid mass of undifferentiated protoplasm, in which some yellowish-brown granules are usually situated. The fibril bundles run longitudinally and show a transverse striation, which is well marked in contracted muscle, though in extended fibres the longitudinal arrangement is more distinct.

The arrangement of the muscular layers is extremely complicated. In the auricles two main layers are apparent, a superficial set of fibres passing from one auricle to the other, and a deeper longitudinal layer which is chiefly confined to each chamber. But both are connected with the septum, and their regular arrangement is disturbed where the great veins enter on the posterior walls.

The arrangement in the ventricles is more complex. The

¹ The structure of the congenital tumours of the heart, rhabdomyomata, supports this theory, for each cell mass contains several nuclei.

walls are much thicker than those of the auricles, but the thickness varies, being minimal at the extreme apex, where it may measure only 2 to 3 mm. ; and maximal in the upper third of the left ventricle, where it may measure 1 cm. The septum is nearly as thick, but the wall of the right ventricle does not exceed 5 mm., and is often thinner. The muscular layers next the endocardium and the pericardium are in a general way arranged longitudinally. The superficial layers run obliquely towards the apex, and then turn suddenly upwards and inwards in a vortex, and ascend on the inner surface. The central bundles are, as a whole, arranged transversely. Some interlace in the septum and form a figure-of-eight loop embracing both ventricles, while others run more or less obliquely from one ventricle to the other, their musculature being thus intimately commingled. In any section of the ventricular wall a few fibres are found to be cut longitudinally and a few transversely, but the vast majority are divided more or less obliquely. The connection of the muscle of the two ventricles is thus very close, and contraction of one side necessarily entails contraction of the other. It is, however, extremely difficult to appreciate the relations of the muscular bundles to the blood vessels, and this can only be followed accurately in the musculi papillares, the cells of which are all arranged longitudinally.

The Connective Tissue in the normal heart is extremely scanty. There are thin loose layers beneath the endocardium and the pericardium, and the two are connected by a fine network, which surrounds the muscle cells and supports the vessels and nerves. But even the main septa are small, and in sections the muscle cells seem everywhere in close apposition. The connective tissue is more abundant at the tips of the musculi papillares where they join the chordæ tendineæ, at the auriculo-ventricular junction, and in the auricular appendices.

The Coronary Circulation.—The heart is supplied with blood by the coronary arteries, and as any interference with their lumen is likely to be accompanied by serious damage to the muscle, their anatomy requires consideration in some detail.

The Right Coronary Artery (Fig. 1) is usually the smaller of the two, and arises from the anterior aortic sinus. It passes to the right in the auriculo-ventricular groove, giving off branches to the adjacent muscle, and terminates in a large branch which runs downwards in the posterior interventricular sulcus. It supplies blood to the right auricle, most of the right ventricle, and the posterior parts of the left ventricle and of the interventricular septum.

The Left Coronary Artery arises from the left posterior aortic sinus, and passes to the left in the auriculo-ventricular groove. It divides almost at once into two main branches, the larger of which descends in the anterior interventricular sulcus, while the smaller continues its course in the auriculo-ventricular groove. They supply the left auricle, the anterior two-thirds of the interventricular septum, most of the left ventricle, and a small portion of the anterior surface of the right ventricle (Fig. 1).

The main horizontal and vertical branches of the two arteries anastomose with each other at their extremities, and thus form around the heart two rings of vessels which are almost at right angles to each other. From these rings smaller arteries penetrate the muscle, giving off still smaller vessels, which end in a very free capillary system, so extensive, indeed, as to surround each individual muscle cell on almost every side. These nutrient arteries, however, differ from the main trunks in that although they anastomose with each other the anastomoses are often, and especially in early life, functionally inadequate to prevent the occurrence of infarction after

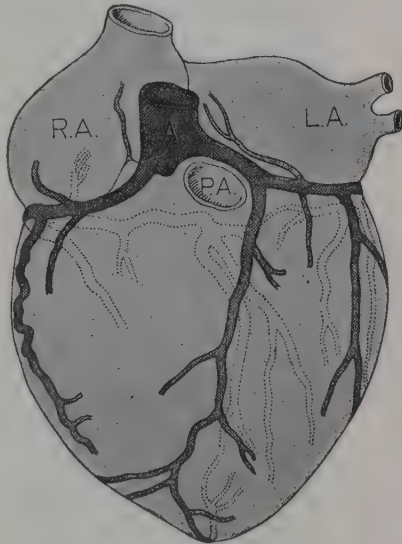


FIG. 1.—THE NORMAL ARTERIAL CIRCULATION OF THE HEART.

R.A., right auricle ; L.A., left auricle ;
A., aorta ; P.A., pulmonary artery.

sudden and complete obstruction of the artery (*see also pp. 42, 362*). In the hearts of relatively old individuals the anastomoses are more ample and patent.¹

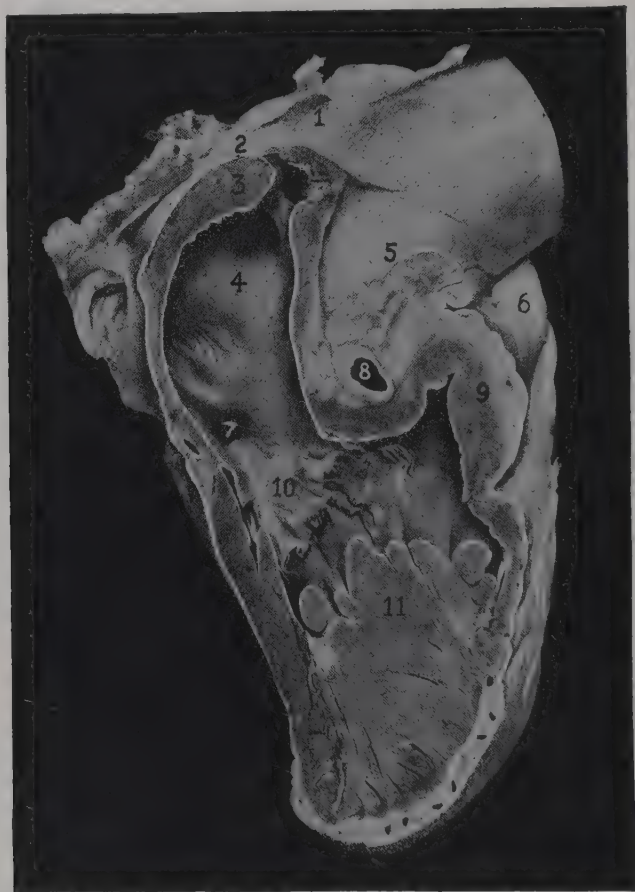


FIG. 2.—THE INTERIOR OF THE HEART VIEWED FROM THE RIGHT SIDE.

1, Superior vena cava; 2, site of the sinus node; 3, tænia terminalis; 4, cavity of right auricle; 5, aorta; 6, pulmonary artery; 7, orifice of coronary sinus; 8, aortic sinus; 9, conus arteriosus; 10, medial cusp of tricuspid valve; 11, interventricular septum.

The coronary arteries send a few branches to the aorta and the pulmonary artery. An anastomosis with corre-

¹ Gross, L., *The Blood Supply to the Heart*, New York, 1921, 77-92.

sponding branches of the bronchial arteries has also been described.

The Nodes, the Bundle and its Branches.—In the embryo the two symmetrical ventral tubes of the hæmocœlum coalesce to form the primitive tubular heart, which subsequently becomes differentiated into its component parts, namely the sinus venosus, auricle, auricular canal, ventricle



FIG. 3.—MICRO-PHOTOGRAPH OF THE SINUS NODE. ($\times 14$.)

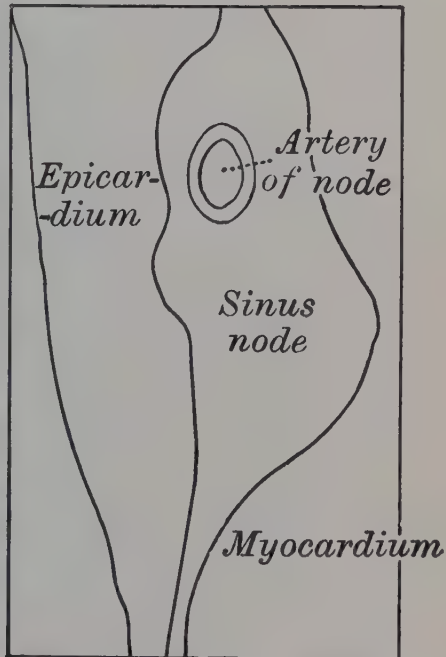


FIG. 4.—SINUS NODE. Diagram of Fig. 3.

and bulbus cordis. In the lower vertebrates, owing to the telescoping of auricle and ventricle upon each other, the auricular canal is represented merely by some circular muscle fibres at the auriculo-ventricular junction, though the sinus venosus and bulbus cordis are still distinct. In the mammalian heart, the sinus venosus is represented by the sinus node, and by some other remnants of primitive tissue near the mouth of the coronary sinus and elsewhere in the auricular wall; the auricular canal is represented by the auriculo-ventricular

node, the bundle and its branches; while the bulbus cordis has become incorporated in the conus arteriosus, its cells being indistinguishable from those of the ventricular muscle.

The *Sinus* or *Sino-auricular Node*¹ (Fig. 3) is situated immediately beneath the epicardium at the junction of the

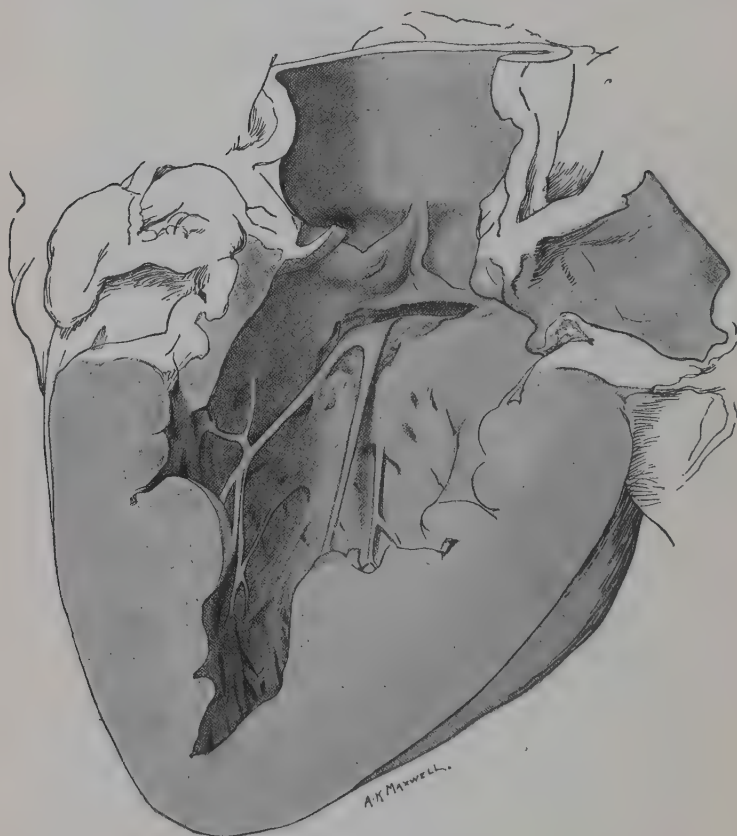


FIG. 5.—HEART OF WALRUS, DISSECTED TO SHOW THE LEFT BRANCH OF THE BUNDLE ON THE LEFT SIDE OF THE INTERVENTRICULAR SEPTUM. (A. KEITH.)

superior vena cava with the right auricle. It measures about 2 mm. in thickness, and extends downwards along the sulcus terminalis for about 2 cm.

¹ Keith, A., and Flack, M., *Journ. Anat. and Physiol.*, 1907, xli., 172.

The Auriculo-ventricular Node ¹ is pear-shaped and measures about 6 mm. longitudinally, and about 3 mm. transversely.

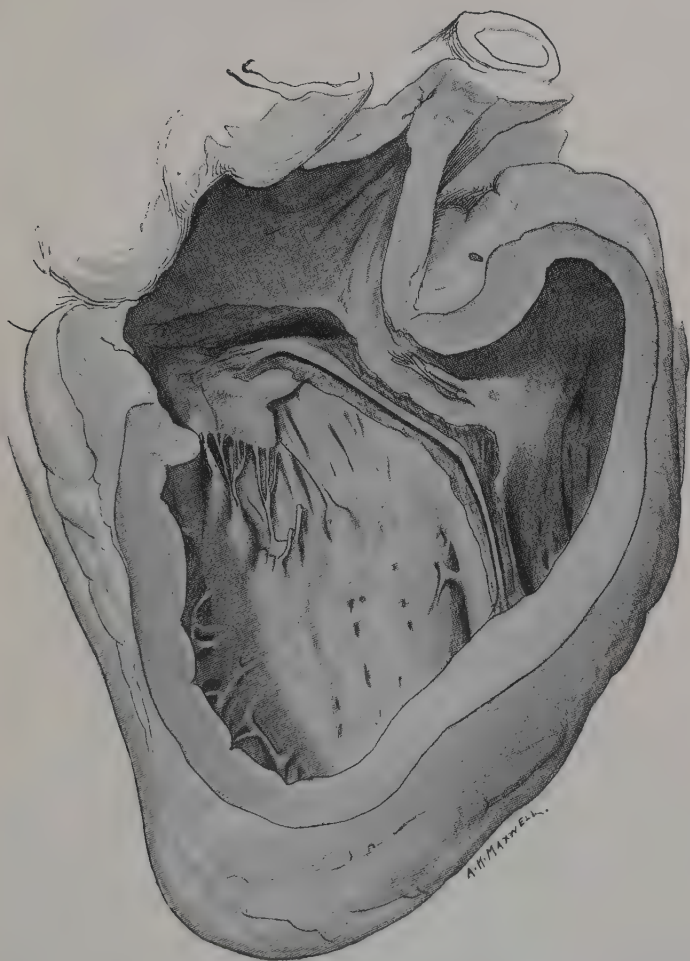


FIG. 6.—HEART OF OX, DISSECTED TO SHOW THE AURICULO-VENTRICULAR NODE AND RIGHT BRANCH OF THE BUNDLE. (A. KEITH.)

It is situated on the right side of the interauricular septum, close to the anterior edge of the coronary sinus and imme-

¹ Tawara, S., *Das Reizleitungssystem des Säugetierherzens*, Jena, 1908. Kent, A. F. S., *Journ. of Physiol.*, 1893, xiv., 233. His, W., Jr., *Arch. a. d. med. Klinik*, Leipz., 1893, 14.

diately above the insertion of the medial cusp of the tricuspid valve. The node consists mainly of muscle fibres which, as they leave the node, are collected together into the *auriculo-ventricular bundle* (Fig. 11). This passes forwards in the interauricular septum and then turns abruptly downwards,



FIG. 7.—HUMAN HEART, DISSECTED TO SHOW THE AURICULO-VENTRICULAR NODE AND RIGHT BRANCH OF THE BUNDLE. (A. KEITH.)

perforating the central fibrous body of the heart, to reach the interventricular septum immediately behind the membranous septum. As it approaches the lower border of the latter the bundle divides into two main branches, which separate and pass downwards on each side of the inter-

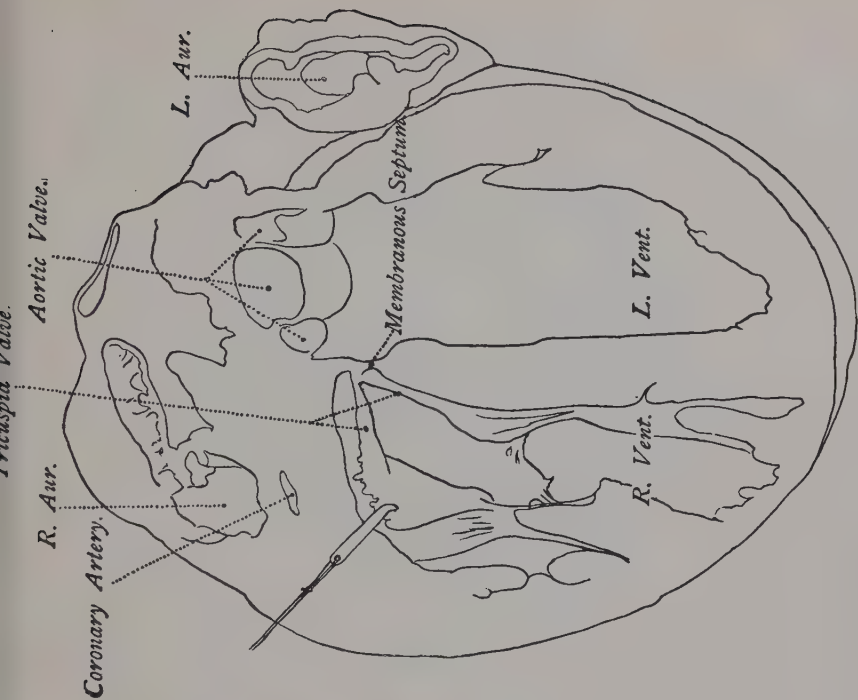


FIG. 8.—LONGITUDINAL SECTION OF THE HEART, SHOWING THE RELATION OF THE MEMBRANOUS SEPTUM TO THE VALVES.

ventricular septum. The right branch is long and slender, and descends far down the right side of the septum before it gives off any secondary branches. The left branch spreads out fanwise soon after it leaves the main stem. The fine terminal arborizations of the branches, the Purkinje fibres, lie in the subendocardial layers of the ventricles and end in the muscle cells, the papillary muscles in particular receiving an ample supply.

As the normal mechanism of the heart's contractions depends on the integrity of the auriculo-ventricular node and bundle, and as these are often involved in lesions spreading from the valves, it is necessary to form a clear conception of the relations of the two. This is best studied by examination of their relations to the membranous septum, as shown in Figs. 8, 9 and 10.

Fig. 8 is a longitudinal section of the heart and demonstrates the insertion of the medial cusp of the tricuspid valve. The aortic cusps are slightly above the membranous septum, and the mitral valve is somewhat posterior. Fig. 9 is a transverse section of the heart through the septum, viewed from below, illustrating another aspect of the relation of the septum to the aortic and mitral valves. Fig. 10 is also a transverse section, but at a higher level, and viewed from above. It is practically in the same plane as the micro-photograph shown in Fig. 11. The orifice of the coronary sinus is shown immediately below the Eustachian valve. To its right is the interauricular septum and the central fibrous body, at the lower margin of which is an aortic cusp, cut transversely, exposing the recesses of one of the aortic sinuses. To its right is a fragment of another aortic cusp, and to its left the membranous septum and the tricuspid valve.

The relations of the auriculo-ventricular node and bundle to the valves are well shown in the micro-photograph (Fig. 11). The plane of the section is not quite horizontal, but slopes downwards from behind. The muscle of the auricle and the insertion of the anterior cusp of the mitral valve (*M*) are visible in the upper part of the photograph, and the section then sweeps forwards along the lower part of the membranous septum, to terminate in the muscle of the interventricular septum. The relations of the tricuspid valve (*T*) to the

posterior part, and of the aortic valve (*A*) to the anterior part of the membranous septum are also shown, as well as the



Aortic Cusp, cut transversely.

Pulmonary Artery.

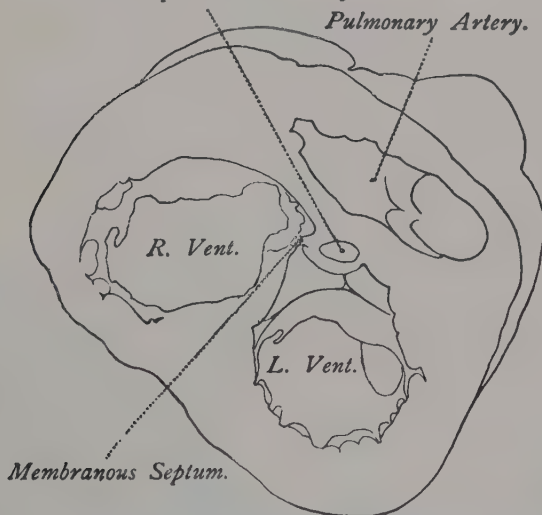


FIG. 9.—TRANSVERSE SECTION OF THE HEART, VIEWED FROM BELOW, SHOWING THE RELATION OF THE MEMBRANOUS SEPTUM TO THE VALVES.

main bulk of the node and almost the whole length of the bundle. The node is most nearly related to the mitral valve, the bundle to the aortic and tricuspid valves.

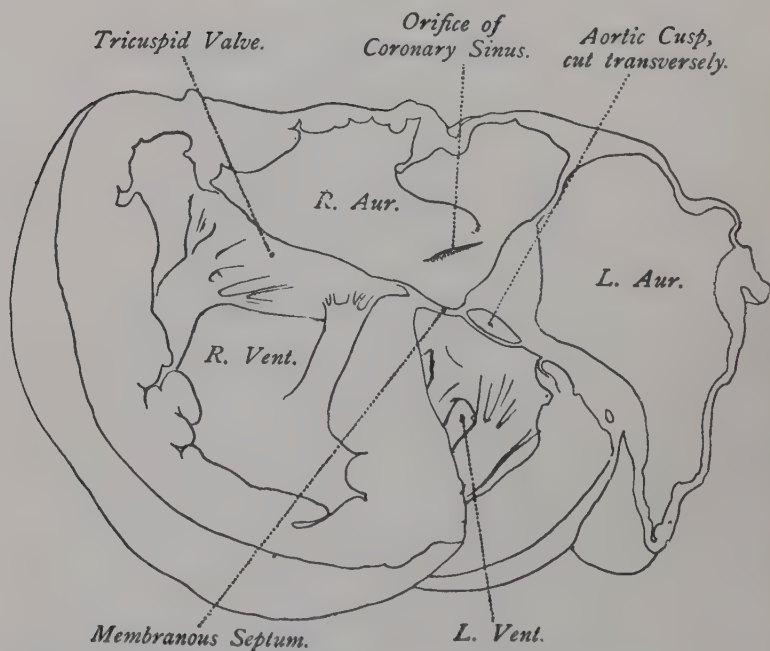
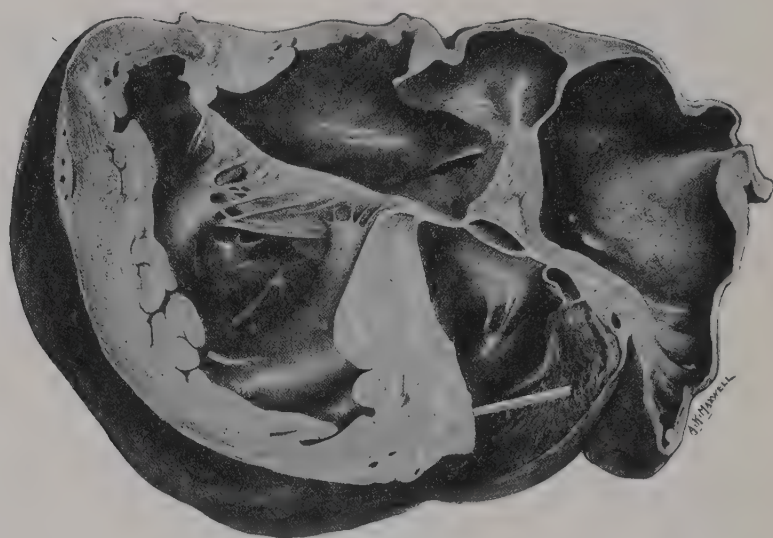


FIG. 10.—TRANSVERSE SECTION OF THE HEART, VIEWED FROM ABOVE, SHOWING THE RELATION OF THE MEMBRANOUS SEPTUM TO THE VALVES.

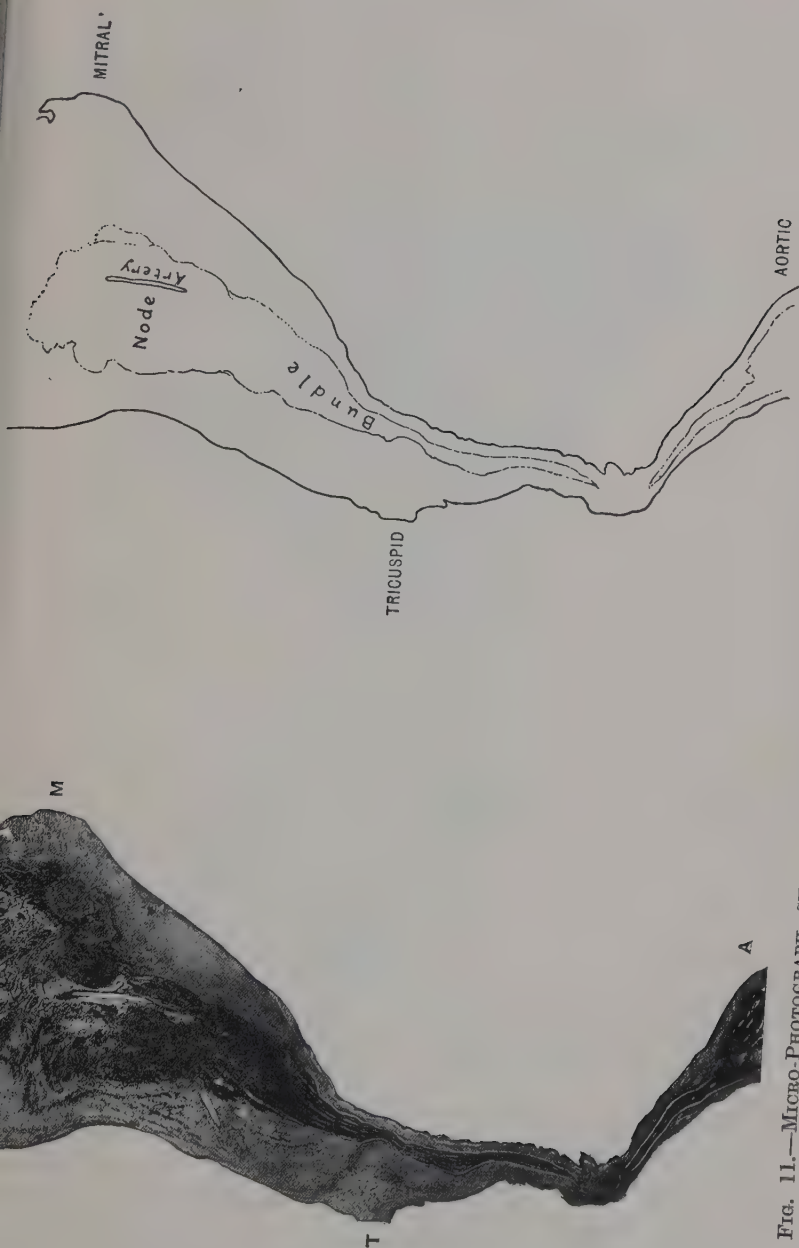


FIG. 11.—MICRO-PHOTOGRAPH, SHOWING THE AURICULO-VENTRICULAR NODE AND ALMOST THE WHOLE OF THE BUNDLE. ($\times 5$.) (A. M. K.)

Fig. 12 represents a similar section. The mitral valve in this case was the site of an acute endocarditis engrafted upon a chronic lesion, and a dense fibrosis can be seen spreading from the base of the valve into the adjacent auricular muscle, and along the membranous septum, which is greatly thickened.



FIG. 12.—MICRO-PHOTOGRAPH OF THE AURICULO-VENTRICULAR NODE AND BUNDLE, SHOWING INFLAMMATORY REACTION AT THE INSERTION OF THE MITRAL VALVE. ($\times 5$.) (A. M. K.)

At the junction of the auricular muscle and the base of the mitral valve there is a well-marked cellular infiltration which has spread into the node. In Fig. 13 the fibrosis is more extensive, and the spread of the fibrosis from the base of the aortic valve into the membranous septum can be seen.

The artery of the node, a branch of the right coronary

artery, is visible in Fig. 11. Many of the lesions which affect the node are due to interference with the lumen of this vessel.

Structure of the Nodes and Bundle.—Both the nodes and the bundle are neuro-muscular. The muscle fibres differ from muscle fibres elsewhere in that they are more slender, fusiform rather than rhomboidal, and striated only at their



FIG. 13.—MICRO-PHOTOGRAPH SHOWING INFLAMMATORY REACTION AT THE INSERTIONS OF THE MITRAL AND AORTIC VALVES. ($\times 5$.) (A. M. K.)

periphery. The nuclei are large and ovoid. The fibres interlace with each other, and are surrounded by connective tissue which is most dense in the sinus node. Both nodes have a well-developed vascular supply. In the sinus node there are many nerve fibres and ganglion cells (Fig. 14), which are directly connected with the vagus and sympathetic nerves.

In the auriculo-ventricular node and bundle of the human heart there are a few nerve fibres, but no ganglion cells, nerve plexus, or nerve endings.¹ The Purkinje fibres, the terminal subendocardial fibres of the bundle, are larger than the fibres of the nodes and bundle.

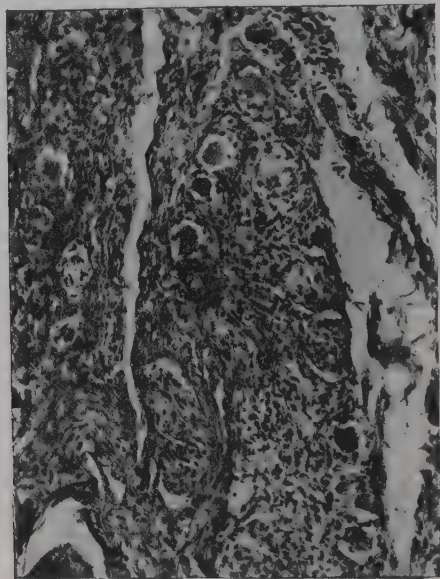


FIG. 14.—MICRO-PHOTOGRAPH OF THE SINUS NODE, SHOWING NODAL TISSUE AND GANGLION CELLS.

The transition from nodal muscle fibre into auricular muscle fibre is gradual. The fibres of the auriculo-ventricular node and bundle are in structural continuity, but there is no proof of direct structural continuity between the two nodes.

¹ Meiklejohn, J., *Journ. Anat. and Physiol.*, 1914, xlviii., 1.

CHAPTER II

PHYSIOLOGY

THE cardiac muscle differs morphologically from voluntary and involuntary muscle elsewhere, and the anatomical differences are associated with differences in physiological action. Stimulation of voluntary muscle is followed, after the latent period, by contraction of the muscle, and this in turn by relaxation. The cardiac muscle also responds to similar stimuli, whether mechanical, chemical, thermal or electric, but there are fundamental differences in the response. During a contraction the heart is refractory to stimuli, and a tetanus cannot, in consequence, be produced. With voluntary muscle the degree of contraction in response to stimulation varies in direct accordance with the intensity of the stimulus, a strong stimulus producing a large contraction, and *vice versa*, for the number of muscle fibres which contract, each to its maximal capacity, varies. In the heart, however, as the muscle fibres are in structural continuity the degree of contraction does not vary with the strength of the stimulus, the cardiac muscle reacting to all stimuli, whether strong or weak, in exactly the same way. A stimulus applied during systole is ineffective because the heart muscle is then wholly refractory. After systole, the refractory period passes away, but immediately after systole the power of contraction is minimal; during diastole it rapidly recovers, and a contraction excited artificially towards the end of diastole is stronger than one excited at an earlier phase of diastole. But the degree of contraction is independent of the degree of stimulation; the cardiac muscle, if excitable, reacting as strongly to a weak stimulus as to a strong one, the "all or nothing" motto of the heart. And, lastly, the heart beats regularly and rhythmic-

ally throughout life, while voluntary muscle is inactive for long periods of time.

THE CONTRACTION OF THE HEART

The current theory of the cardiac action is in some respects a revival of an old hypothesis. It was at first supposed that the contractions were due to the direct action of the blood as it flowed into the auricles, occurring as soon as the chambers were sufficiently distended. But it was soon shown that this could not be the case, as the frog's heart, when separated from the body and empty of blood, would beat rhythmically for a considerable time if placed under favourable conditions of moisture and warmth. Its rhythmic action was therefore ascribed to an inherent property of the myocardium. The discovery of nerve cells and fibres in the heart then followed, and, as respiration was at this time considered to be due to the automatic action of nerve cells, the cardiac contractions were ascribed to a similar cause. The myogenic theory was, however, again reverted to when Gaskell showed that the isolated apex of the heart, which is devoid of nerve cells, may contract rhythmically for long periods. In consequence the cardiac beat must be due to an inherent power of the muscle to produce rhythmic stimuli.

The neurogenic theory, however, still finds supporters, who claim Carlson's observations on the heart of the king crab, *Limulus*, as proof of the nervous origin of the heart's contractions. But although the heart of *Limulus* ceases to beat after removal of its median nerve cord, its response to stimulation is like that of skeletal, not like that of cardiac, muscle.

The wave of contraction, starting at the junction of the superior vena cava and right auricle, travels through auricle to ventricle, and a controversy arose as to whether the stimulus was conducted by nerve or by muscle. Gaskell showed that in the tortoise the conduction must be by muscle because the coronary nerve, the sole nervous connection between auricle and ventricle in this animal, lies outside the heart, and can be severed without disturbing the normal sequence of contraction; whereas section of the muscle alone leads to

asynchronous contractions of the chambers. Gaskell's conclusions were at first alleged to be inconclusive, and inapplicable to mammalian hearts, as no muscular connection between auricle and ventricle was at that time known. But Stanley Kent¹ and His² soon afterwards demonstrated the connecting fibres, the auriculo-ventricular bundle, which we now know to be a neuro-muscular structure (*see* p. 16).

THE PROPERTIES OF THE CARDIAC MUSCLE

The heart muscle possesses other properties besides that of generating stimuli for contraction. It has the property of receiving stimuli, *excitability*; of passing into contraction in response to a stimulus, *contractility*; of conducting stimuli throughout its substance, *conductivity*; of offering resistance to stretching, *tone* or *tonicity*.

These various functions are not equally well developed in every part of the mammalian heart. In the primitive tubular heart stimulus formation and contractile force are fairly equally developed throughout, and a contraction can originate in any part of its course. But in the vertebrates the musculature of the auricle and ventricle has developed its contractile power, and in large measure lost its power of originating contractions, responding with a single beat to an experimental stimulus. In the frog, however, the parts which remain least altered, the sinus, auricular canal and bulbus cordis, have a rhythmic power which is greatly in excess of that possessed by auricles and ventricle, readily initiating contractions spontaneously, and responding to a minimal stimulus, not with a single isolated contraction, but with a series of beats.

In the mammalian heart stimulus production is most active in the sinus node, stimulus conduction is most manifest in the bundle and its branches, and contractility in the muscle cells of the auricles and ventricles.

In the mammalian heart the nodes, the bundle and its branches possess the inherent power of generating rhythmic stimuli for contraction. The exact nature of the process

¹ Kent, A. F. S., *Journ. of Physiol.*, 1893, xiv., 233.

² His, W., Jr., *Arch. a. d. med. Klinik*, Leipz., 1893, 14.

whereby a stimulus is elaborated is still obscure. But we know that in the normal heart the rate of stimulus formation and discharge is greater in the sinus node than elsewhere. In the human heart the rate of stimulus formation in the sinus node is about 70 per minute, in the auriculo-ventricular node probably about 50, and in the bundle about 30. In health the sinus node, having the most frequent rate of impulse formation, sets the pace of the whole heart, and in health no beats originate elsewhere. When the normal heart contracts the first part to become electro-negative is the sinus node, and an extrasystole, produced experimentally by stimulation of this area, yields an electrical response exactly similar to that of the normal beat.

The stimulus is transmitted from the sinus node directly to the auricular muscle fibres and, as the muscle fibres of the auricles are in structural continuity, a synchronous contraction of both auricles ensues. The stimulus is also transmitted from the sinus node, by paths which have not yet been fully defined, to the auriculo-ventricular node, and thence through the bundle and its branches to the muscle fibres of the ventricles, which, on receiving the stimulus, contract simultaneously. The response of the heart to each sinus stimulus is therefore universal, the auricles and ventricles contracting in regular sequence about 70 times a minute. The heart beating in this way is said to have a sinus rhythm.

The power of generating impulses is normally latent in the auriculo-ventricular node, in the bundle and in its branches, on account of the more frequent rate of stimulus formation in the sinus node. But, in disease or in animal experiments, the rate of stimulus formation in the former sites may be increased, and contractions may then arise therein. These ectopic or heterotopic contractions may arise if the rate of stimulus formation in the sinus node is lowered below that of the auriculo-ventricular node or elsewhere, or if the rate of stimulus formation in any part of the heart rises above that of the sinus node. The heart has then no longer a sinus rhythm, the disturbed mechanism being spoken of as nodal (auriculo-ventricular) rhythm, ventricular tachycardia, etc. In other cases the sinus rhythm is preserved, but interrupted from time to time by ectopic contractions originating in other

parts of the heart than the sinus node. These beats, which are termed extrasystoles, are discussed in Chapter VI.

The regular auricle-ventricle sequence of the heart's contractions, dependent as already described on the regular formation of stimuli in the sinus node, is also dependent upon the integrity of the pathways which transmit the stimuli through the heart, and especially upon the integrity of the auriculo-ventricular bundle and its branches. If the bundle is severed, experimentally or by disease, the regular auricle-ventricle sequence is dissociated, the auricles and ventricles contracting irrespective of each other and at a totally different rate. The auricles, still receiving stimuli from the sinus node, contract about 70 times a minute, while the ventricles, failing to receive sinus stimuli, contract about 30 times a minute in response to stimuli arising in the bundle below the point of severance (*see* p. 121).

THE REGULATION OF THE ACTION OF THE HEART

The Nervous Control of the Heart

The rate and force of the cardiac contractions are subject to great variations even in health, those due to nervous influences being perhaps the most conspicuous. The forcible, frequent cardiac action induced by emotional stress is familiar to all, and although the heart is not under the direct control of the will, its action may be modified profoundly by the action of both components of the autonomic nervous system, namely the sympathetic system, and the parasympathetic, represented by the vagus. Their action has been likened to the spur and the bridle, the former increasing and the latter decreasing the speed and the force of the cardiac contractions.

The Action of the Vagus.—Both experimentally and in the human heart the vagus normally exerts an inhibitory influence, restraining the rate of stimulus formation in the sinus node. This inhibition, constituting the normal vagal tone, may be increased by excitation of the vagus nerve. In man this may be effected by direct compression of the nerve on either side of the neck, though the right vagus is usually the more effective; by reflex stimulation, as by compression

of the eyeball ; or by the administration of pilocarpine, caffeine, or drugs of the digitalis group. Vagal tone can be abolished experimentally by section of the nerves ; and in the human heart by the administration of atropine which paralyses the post-ganglionic fibres in the heart itself. The subcutaneous injection of one-thirtieth of a grain of this drug causes a transient paralysis which reaches its maximum in twenty to thirty minutes and then gradually subsides.

Vagus action retards the rate of stimulus production in the sinus node, and thus retards the rate of the whole heart (Fig. 15). Increase of vagal tone slows the rate of the cardiac contractions (sinus bradycardia), whereas lessening of vagal tone accelerates the pulse (sinus tachycardia, p. 78). Increase

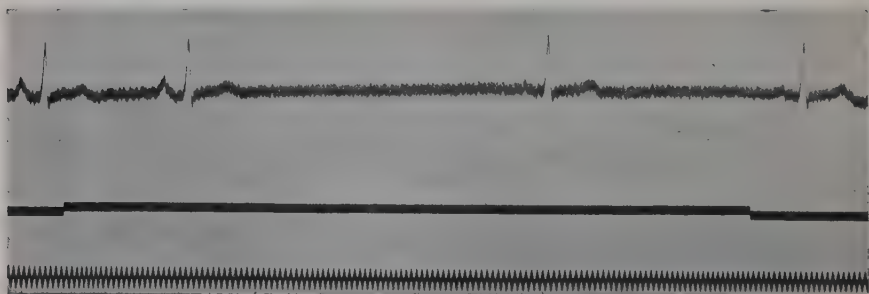


FIG. 15.—VAGAL INHIBITION.

Compression of the right vagus during the period shown by the signal retards the whole heart and lessens the amplitude of the auricular deflexion. Derivation II.

of vagal tone also weakens the contractility of the auricular muscle, lowers its excitability, and impedes or blocks the transmission of stimuli within it, causing sino-auricular block (*see* p. 140) and auricular fibrillation (*see* p. 175). These effects of vagal stimulation are well known in animal experiments and have also been observed in the human heart.¹ Moreover, there is some evidence which suggests that increased vagal tone lowers the tonicity of the auricular muscle.

The effects of increased vagal tone upon the auriculo-ventricular node and bundle are also pronounced, and are shown by depression of conductivity. Mild stimulation delays the transmission of the stimulus from auricle to ventricle,

¹ Ritchie, W. T., *Quart. Journ. of Med.*, 1912-13, vi., 47.

the *P-R* interval in the electrocardiogram being lengthened. Stronger stimulation blocks the transmission of an occasional impulse, or may even prevent every stimulus from reaching the ventricles. These facts are of fundamental importance in relation to functional auriculo-ventricular block, and the mode of action of drugs of the digitalis group.

The vagi have less influence upon the branches of the bundle than upon the bundle itself or the nodes.

The Vagi have no direct action upon the Ventricles.—This is clearly shown in complete auriculo-ventricular block (see p. 115). When the heart beats with a sinus rhythm the vagal influence upon the ventricles is indirect, the retardation of the whole heart lengthening ventricular diastole and thereby increasing the contractility of the ventricular muscle. The pulse is stronger because it is less frequent.

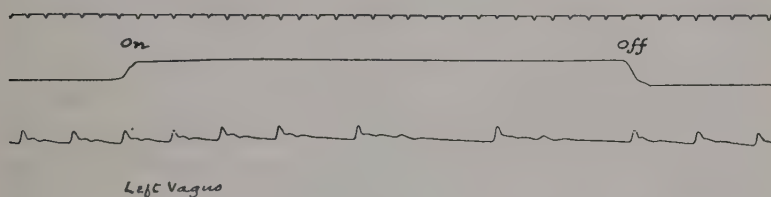


FIG. 16.

The upper curve shows the period of pressure upon the left vagus nerve. Diastole is lengthened by this procedure, and extra-systoles occur in consequence.

In health the vagal tone is lessened during inspiration, and increased during expiration, thus causing the physiological, or respiratory, irregularity of the pulse which is so frequent in young persons that it has been termed the juvenile form of cardiac irregularity (see p. 84). Lowered vagal tone is one of the factors which lead to more frequent cardiac action during physical exercise. Increased vagal tone (vagotonia), revealed by excessive response to pilocarpine which stimulates the vagus, by greater tolerance for atropine which paralyses the vagus, and for adrenalin which stimulates the sympathetic, is probably due to the action of hormones, though their activity is still largely hypothetical.

The Action of the Sympathetic Nerves.—The action of the sympathetic nerves, in contrast to that of the vagus, leads

to increased frequency and force of the cardiac contractions. Sympathetic stimulation occurs during emotional stress, during physical exercise, and following the administration of adrenalin. It may also result from excess, or abnormality, of the thyroid or other internal secretions, leading to an increased discharge of adrenalin into the blood (hyperthyroidism). The most striking clinical manifestation of overaction of the sympathetic (sympatheticotonia), is shown in the sinus tachycardia of exophthalmic goitre and other forms of hyperthyroidism, and in "nervous" individuals, whose hyper-sensitivity may be demonstrated by the excessive response to the injection of adrenalin chloride solution into the veins. In these individuals the rate and the force of the cardiac contractions are constantly much greater than the requirements of the tissues for arterial blood necessitate, so that much of the work of the heart is misspent.

The Mechanical Control of the Heart

The chief function of the heart is to maintain the flow of blood and thus keep the arterial pressure at such a height that the varying needs of the organs and tissues of the body are met by an adequate supply of blood, and thus by a sufficiency of nutriment and oxygen. In a healthy adult man the output per beat is calculated to be about 120 c.c., and the output per minute (minute volume) to be 5 to 8 litres at rest.¹ When the demand of the organs and tissues for oxygen increases during physical exercise, the heart responds by beating more frequently, thus increasing its minute volume. At the same time the beats become more forcible. During strenuous physical exercise, when the needs of the tissues are greatest, the minute volume may reach 24 litres. The daily work of the two ventricles is said to be sufficient to raise a man of average weight to "about twice the height of the highest skyscraper in New York."²

How does the heart increase its output during physical exercise? We know that the beats become more frequent.

¹ Douglas, C. G., and Haldane, J. S., *Journ. of Phys.*, 1922, lvi., 69.

² Macleod, J. J. R., *Physiology and Biochemistry in Modern Medicine*, Lond., 1919, 213.

The contractions may also be more forcible ; but although in some observations the output per beat is raised, in others it is certainly constant, being no greater during physical exercise than at rest. If the output per beat is increased this may be due to a physiological dilatation of the chambers as a result of the greater venous inflow from the active voluntary muscles, and of the greater venous inflow produced by the deeper and more frequent breathing induced by the exercise. The hypothesis that the negative pressure within the chambers early in diastole is an important factor in promoting the venous inflow is now discarded. The physiological dilatation of the heart stretches the muscle fibres to their optimum length, and increases their contractile force ; for it has been shown that the heart, like the other muscles, develops energy on contraction, in direct proportion to the length of its fibres.¹ The contractions become stronger, and although the venous inflow is greater there is no increase of residual blood, because the heart beats more rapidly and at the same time the output per beat may be greater in some instances.

The increased frequency of the contractions, which must be regarded as the main factor producing a greater minute volume, is brought about by lessening of the vagal tone and increase of the sympathetic tone, which, acting on the sinus node, accelerate the formation of the stimuli. An increased output may therefore be due to (1) increased venous inflow, (2) lessened vagal tone, and (3) increased sympathetic tone. It is also believed to be the result of an increased hydrogen ion concentration of the blood (*see* p. 27). During physical exercise all four factors are in action.²

The Reserve Power of the Heart.—The reserve power of the heart is its capacity for increasing its output according to the needs of the body during physical exertion. In health the reserve power is large. During physical exertion, short of the most strenuous, the heart, beating more forcibly and more

¹ Starling, E. H., *Linacre Lecture on the Law of the Heart*, Lond., 1918.

² Bainbridge, F. A., *The Physiology of Muscular Exercise*, Lond., 1919.

rapidly, meets, by an increased and adequate output, the demand of the tissues generally, and of the heart itself, for more oxygen, and the work is done without distress or shortness of breath. If the contractility of the heart is lowered by toxæmia, anoxæmia, or inflammation, if its blood supply is lessened by disease of the coronary arteries, if the normal auricle-ventricle sequence of contraction is disturbed, or if the valves are diseased, the output may still be sufficiently large to meet the demands of the tissues while the patient is at rest. But in this case, most notably in aortic and in mitral incompetence, the work performed is greater than in health and the reserve power is in consequence lessened. The increased work of the heart produces hypertrophy, or dilatation and hypertrophy, of the chambers, and these changes are an essential factor in the "compensation" of valvular disease; and if the cardiac output, while the patient is at rest, is sufficient, the breathing is easy and quiet.

On physical exertion, however, in such a patient the output per beat of the enfeebled or dilated heart may be insufficient to meet the increased demands of the tissues, and the minute volume has to be raised by undue acceleration of the cardiac rate of contraction. The pulse therefore becomes more frequent than the pulse of a man with a healthy heart who is undertaking the same amount of exertion. And even although the rate be much accelerated, even to 150 or 160 per minute, the output may be insufficient to meet the demands of the tissues, and shortness of breath ensues.

If the reserve power is still further reduced, the patient may be breathless even when at rest, and the breathlessness will be intensified by even the least physical exertion. In auricular flutter and other forms of extreme tachycardia, the rapid rate of contraction probably entails a lessened output per beat, and this in turn causes acceleration, a vicious circle being thus established.

That the reserve power of the heart depends not merely upon an increased output per beat, but also upon a greater frequency of the contractions, is demonstrated by patients with complete auriculo-ventricular block. In these patients the ventricular rate remains constant, usually about 30 per minute, and does not become accelerated during physical

exercise, and the patient's capacity for exercise without dyspnoea is therefore very small.

The increased output of a normal heart during physical exercise furnishes a more ample and a more rapid flow of blood to the muscles, the brain, and the heart itself. The splanchnic vessels become narrowed, and the blood pressure in the systemic and coronary arteries rises. This rise of the blood pressure is due to stimulation of the sympathetic nerves, and we know that adrenalin, which is discharged into the blood during emotional stress, causes systemic vaso-constriction, coronary dilatation, and increased force of the cardiac contractions. It has therefore been suggested that these changes when occurring during physical exercise are likewise the expression of an increased discharge of adrenalin into the blood, but this hypothesis still lacks proof.

Dyspnoea.—During physical exercise the consumption of oxygen and the output of CO_2 by the muscles are increased in accordance with their activity. This demand for more oxygen and for the elimination of the excess of CO_2 is met by a compensatory activity of the circulatory and respiratory mechanisms, but if either fails to meet the demand and the breathing becomes laboured, breathlessness or dyspnoea occurs.

The normal stimulus to the respiratory centre is the reaction of the blood, or its hydrogen ion concentration (C_H), the value of which is usually expressed as P_H . In a neutral solution the concentration of hydrogen ions, H ions, and of hydroxyl ions, OH ions, is equal, whereas H ions are in excess in acid solutions, and OH ions in alkaline solutions. In health the C_H of the blood, both arterial and venous, is practically constant, because it contains buffer salts which constitute a reserve of alkali, of which a portion is available for the neutralization of H_2CO_3 or other acids which may enter the blood.

In the blood the CO_2 occurs as H_2CO_3 or is bound to bases to form bicarbonate. The constancy of the C_H of the blood is dependent on a constant ratio between the CO_2 as H_2CO_3 and CO_2 as bicarbonate. This ratio is normally 1 to 20. The CO_2 of the blood is carried by the plasma and by the alkali of the hæmoglobin. The latter contains the chief reserve of

alkali. The hæmoglobin parts with much of its alkali when oxyhæmoglobin is converted, by loss of oxygen, into reduced hæmoglobin; and CO_2 on entering the blood is neutralized by being bound to this alkali to form bicarbonate. When the CO_2 leaves the blood, the hæmoglobin combines again with its alkali. Thus the C_H of the blood is maintained remarkably constant.¹ If the CO_2 tension rises still further, the C_H of the blood is thereby increased, the respiratory centre is stimulated, and the more ample pulmonary ventilation which ensues leads to the elimination of CO_2 until the ratio of $\text{H}_2\text{CO}_3 : \text{NaHCO}_3$ again falls to 1 : 20.

Any lowering of the alkali reserve, lessening the alkalinity of the blood and constituting the condition known as acidosis, might conceivably be an important factor in the causation of dyspnœa, because the respiratory centre is stimulated by any increase in the C_H of the blood. Deficiency of the alkali reserve is seldom due to excessive production of acid. But we know that physical exercise in conjunction with an inadequate supply, and especially a prolonged deficiency, of oxygen, leads to the appearance of lactic acid in the blood. In disease, notably in diabetes mellitus and in starvation, acid production may be excessive, and severe dyspnœa may ensue as a direct manifestation of the acidosis.

The dyspnœa of heart failure, however, is not associated with acidosis, for the alkali reserve of the blood plasma, estimated by Van Slyke's² or other method, is not lowered provided that the renal functions are unaffected. The same is true of the dyspnœa complained of by those who present the so-called D.A.H. Defective elimination of acids, however, lowers the alkali reserve. This is observed in renal disease and in those forms of it which are so frequently associated with disease of the heart. The dyspnœa of these patients, although essentially anoxæmic in nature, is toxic in origin, is often paroxysmal in character, perhaps cyclic, often nocturnal in its incidence, and not related to exertion. The dyspnœa of acidosis is thus usually sufficiently characteristic to differentiate it clearly from the more common dyspnœa of cardiac origin (*see* p. 280). When dyspnœa occurs in the

¹ Van Slyke, D. D., *Physiol. Reviews*, 1921, i., 141.

² Van Slyke, D. D., *Journ. Biol. Chem.*, 1917, xxx., 347.

terminal stages of cardiac disease while the patient is at rest, the alkali reserve may be lowered, probably as the result of defective renal elimination. In some instances, however, the dyspnoea is associated with alkalosis, not acidosis.¹

Respiratory Exchange.—In a normal individual at rest, breathing 6 to 8 litres of air per minute, the blood while passing through the lungs becomes almost fully saturated with oxygen. The oxygen unsaturation, namely the difference between the oxygen content and the total oxygen capacity of the hæmoglobin, is on the average less than 5 per cent. According to Harrop² the oxygen saturation of arterial blood varies between 94 and 100 per cent., the average being 95·5 per cent. According to Meakins and Davies³ the average is 95·3 per cent., and they find that the average normal CO₂ content of arterial blood is 52·7 volumes per cent. While passing through the systemic capillaries the blood parts with some of its oxygen, and takes up some CO₂. The oxygen saturation of venous blood taken from a vein of the arm varies within wide limits, the normal 56·4 per cent. falling to zero when the arm is cooled and rising to 94·2 per cent. when the part is heated.³ Observations on the oxygen and CO₂ content of blood which has been withdrawn from superficial veins must therefore be accepted with considerable reserve. In cardiac patients without arrhythmia or signs of heart failure, who were resting, Harrop found no deviation from the normal values of oxygen and CO₂ in the arterial and venous blood, whereas in most of the patients with heart failure and signs of pulmonary congestion the oxygen unsaturation of the arterial blood was abnormally high. In another series of cardiac patients without cardiac failure Lundsgaard⁴ determined the oxygen unsaturation of the venous blood and found it to be within normal limits. In patients with heart failure the unsaturation was above the normal. In auricular fibrillation and in "D.A.H." the oxygen unsaturation of the venous

¹ Fraser, F. R., Ross, J. P., and Dreyer, N. B., *Quart. Journ. of Med.*, 1922, xv., 195.

² Harrop, G. A., *Journ. of Exper. Med.*, 1919, xxx., 241.

³ Meakins, J., and Davies, H. W., *Journ. of Path. and Bact.*, 1920, xxiii., 451.

⁴ Lundsgaard, C., *Ibid.*, 1918, xxvii., 179, 199, 219.

blood may vary considerably. In auricular fibrillation it may be high, even in the absence of symptoms of failure.

The volume of air breathed during moderate physical exercise rises to about 20 to 30 litres per minute because of the call of the tissues for oxygen. The excess CO_2 added to the venous blood must also be eliminated, and the increased tension of CO_2 raises the C_H of the blood and stimulates the respiratory centre, whose excitability is raised under the influence of the higher centres. The greater pulmonary ventilation which ensues increases the expansion of, and the residual air in, the lungs and promotes the diffusion of oxygen from the alveolar air into the blood, and of CO_2 from the blood into the alveolar air. The oxygen tension of the blood is thus kept as high, and the CO_2 tension as low, as when the individual is resting.

In heart failure the anoxæmia, or increased oxygen unsaturation of the arterial blood, may be a result of the lowered reserve power of the heart, of secondary pulmonary lesions such as congestion and œdema, or of both factors, acting concurrently and constituting a vicious circle. Moreover, an abnormally high oxygen unsaturation of the venous blood may depend not only upon a pronounced unsaturation of the arterial blood, but also upon an undue retardation of the blood flow through the capillaries, whereby the oxygen unsaturation is still further increased.

Dyspnœa is intensified if the blood, because of anæmia, is deficient as an oxygen carrier and CO_2 carrier. The cyanosis of heart disease, indicating pronounced oxygen unsaturation of the capillary blood, is usually accompanied by polycythæmia, which is, as suggested by Gibson,¹ a compensatory change due to the lessened activity of the red cells as oxygen carriers, to their lessened wear and tear, and the consequent longer duration of their individual existence. The dyspnœa of heart failure, too, may be aggravated if the pulmonary expansion is defective from compression of the lung by hydrothorax, aneurysm, flatulent distension of the abdomen, etc., or from disease of the lung itself.

¹ Gibson, G. A., *Proc. Royal Soc. of Edin.*, 1903, xxiv., 393.

CHAPTER III

THE DISEASES OF THE MYOCARDIUM

Hypertrophy—Atrophy.—The muscle cells in the normal heart vary considerably in size, those on the left side being appreciably larger than those on the right, and the cells in the auricles smaller than those in the ventricles. There is

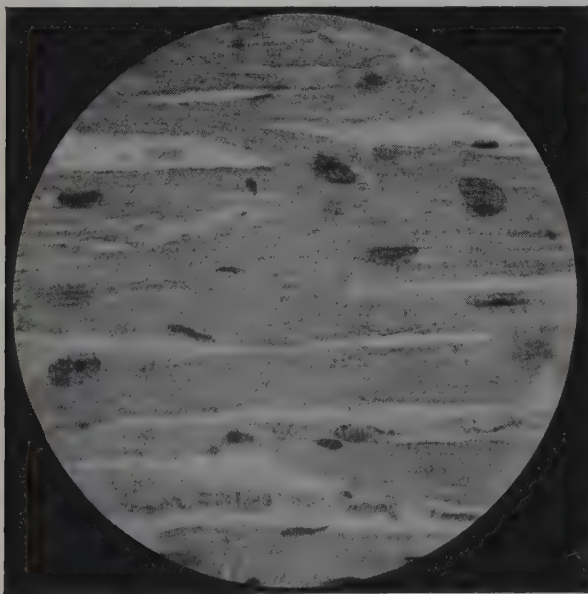


FIG. 17.—HYPERTROPHY OF MUSCLE FIBRES. ($\times 400$.)

often considerable difference between even adjacent cells, and it may be difficult to estimate the average size in any particular case. It is, however, always greater than normal in hypertrophied hearts (Fig. 17), the increase being chiefly in the transverse diameter, and in other respects the cells seem

normal. There has been considerable discussion as to whether the cells are more numerous as well as larger. Most observers are against any numerical increase, but Heller and Zielonko believed that they had found evidence of regeneration. The connective tissues, too, in hypertrophied hearts are always increased in amount.

In *atrophied* hearts the cells are smaller than usual (Fig. 18). In some cases no other abnormality can be detected, but in others the perinuclear pigment is excessive. The pigment

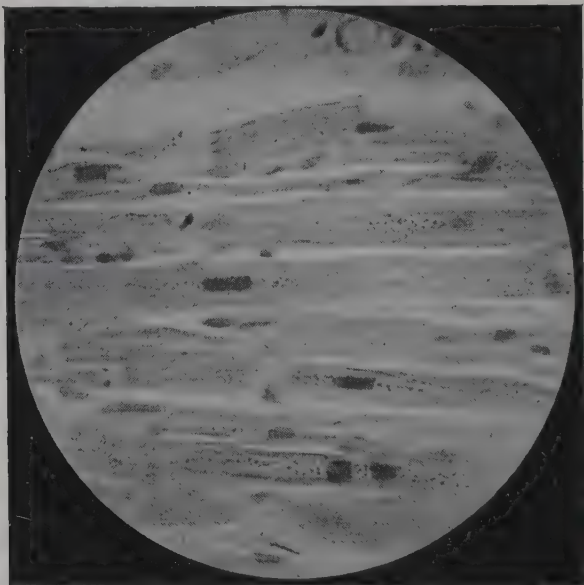


FIG. 18.—ATROPHY OF MUSCLE FIBRES, SHOWING INCREASE OF PERINUCLEAR PIGMENT. ($\times 400$.)

granules appear in the normal heart about the age of ten years, and their number increases with advancing years. They are usually numerous in senile hearts. They are brownish yellow in colour and of varying size, and are said to be composed of hæmatoidin. They lie in conical masses at each pole of the nucleus in the perinuclear space. In atrophy they may be numerous or scanty.

Ischæmic Atrophy.—In cases of local starvation from obstruction of the coronary arteries other changes may be

noticed in the cells. Those around the arterioles are but little smaller than normal, but those a little distance away are distinctly diminutive. They are, too, empty in appearance, the central fibril bundles having disappeared, leaving only a few at the periphery of the cell (Fig. 19). The perinuclear mass is large and stains faintly, and the lesion is readily appreciated in both longitudinal and transverse sections. The nucleus is always abnormal, and is at first large with a scanty chromatin network, which may be here and there aggregated into little clumps; while in the later stages when the cell is tiny, the nucleus becomes minute and pyknotic.

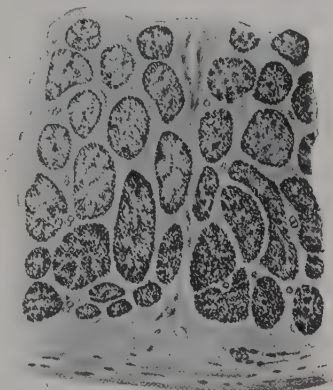


FIG. 19.—ISCHEMIC ATROPHY.
($\times 120$.)

The cells nearest to the thickened endocardium are least altered.

Granular Degeneration.—Granular degeneration (cloudy swelling) is in some ways comparable to ischæmic atrophy. In the early stages the proper structure of the cell is obscured by coarse granules which, scattered widespread through the cell, are most numerous in the central part where the striation is almost lost. In the later stages (Fig. 20) the cells are smaller and the perinuclear mass

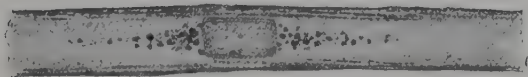


FIG. 20.—GRANULAR DEGENERATION IN AN ADVANCED STAGE. ($\times 1000$.)

enlarged, only a few fibrils with poorly marked striation persisting at the periphery. Still later the striation disappears, leaving a granular mass of an irregular oval shape. In ischæmic atrophy the fibrils show the transverse striation so long as the muscle cells are recognizable. In granular degeneration the striation is obscured at an early period, before atrophy commences.

Granular degeneration occurs in all the acute infections, and is more extreme the longer the illness. It is often accompanied by some fatty and hyaline change.

Hyaline Degeneration.—Hyaline degeneration is relatively uncommon. The affected cells are homogeneous and glistening in appearance, all the characteristic structure having disappeared. The staining reactions are altered and the cells colour yellow instead of red-brown with Ehrlich's triple stain. The distribution of the change varies. Sometimes only a part of a cell is affected, but more often a whole cell or a group of contiguous cells. As a rule the area involved is small, but it may be large and widespread.

The change is due to coagulation necrosis, and is always present in recent infarcts. It is also produced by toxic causes, as it is seen in the vicinity of inflammatory foci, and may be found in a few cells in the acute infections.

Fatty Infiltration.—Some fat is always present in the sub-pericardial connective tissue, though the amount varies in different cases, being sometimes scanty and sometimes so great that it covers and obscures the muscle. Normally it is thickest in the auriculo-ventricular sulcus and over the right heart.

Under pathological conditions the fat may infiltrate the muscle, spreading along the main fibrous septa, in particular around the blood vessels, and even between individual muscle fibres (Fig. 21). As a rule the infiltration is superficial, but in some cases it penetrates deeply and may even appear in little patches beneath the endocardium. Infiltration is always present whenever the superficial fat is excessive, but it may be extreme without notable excess of the superficial fat. It is always greatest on the right side, where it may be considerable though the left side be but little affected.

In normal hearts one or two layers of muscle cells in the immediate vicinity of the superficial fat generally show some fatty degeneration, but the change is strictly limited. In fatty infiltration a similar condition obtains, but to a greater extent, and a majority of the cells may be involved.

Fatty infiltration is usually merely a part of general obesity.

In exceptional cases, whose nature is not understood, however, the heart alone may be affected.

Fatty Degeneration.—Fatty infiltration is readily appreciated on naked-eye examination, but fatty degeneration often can only be recognized microscopically, even in cases where it is well marked; for the colour changes by which its presence is shown may be obscured by congestion of the vessels or jaundice, and in hypertrophy and atrophy. Special staining methods, too, are required for its appreciation, as in the usual methods the fat is dissolved out of the cells and, although the honeycomb appearance in extreme cases is

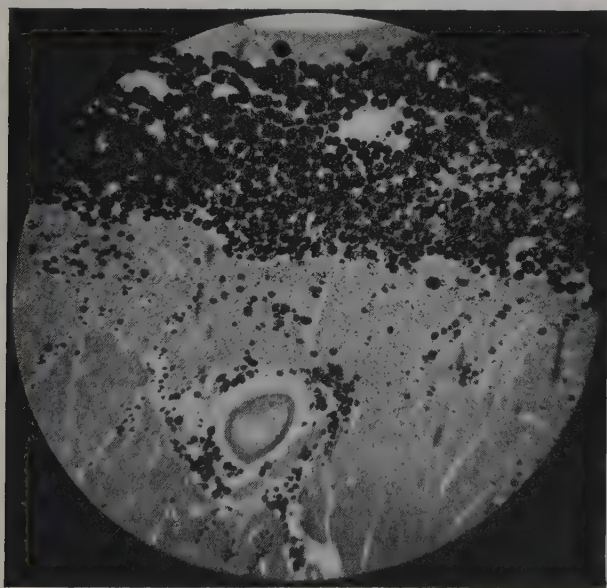


FIG. 21.—FATTY INFILTRATION OF MUSCLE IN THE VICINITY OF THE PERICARDIAL FAT. STAINED WITH OSMIC ACID.

quite distinctive, the lesser degrees are readily missed. It is extremely common, but the frequency of its occurrence is considerably under-estimated on account of the failure to employ the special technique required for its detection.

In a typical case the appearance of the heart is very characteristic, little spots of buff or yellow colour being

scattered over the endocardium. The mottling is sometimes very fine and more or less diffuse, but more commonly the spots are massed together, forming bands which, on the papillary muscles, run transversely. These bands are irregular and often zigzag in contour, and when distinct, are aptly compared to the markings on a thrush's breast. The change is usually apparent only beneath the endocardium, and on section is seen to penetrate for but a short distance into the muscle. But occasionally it involves the whole wall, and it may even be visible beneath the pericardium. Very rarely it is apparent only on the pericardial surface.

The whole of the heart is never equally involved, and most frequently only a part is affected. It is most common in the left ventricle, and it occurs but rarely in the auricles. One ventricle may be extensively diseased, and the other but slightly. In the left ventricle the papillary muscles, the adjacent muscle on the posterior wall and the septum are most often degenerate; on the right side the papillary muscles and the muscle in their vicinity. The left ventricle suffers alone, or to the greatest extent, in anæmia and aortic valvular disease. In chronic pulmonary disease and in mitral affections the right ventricle is chiefly involved.

On microscopic examination of sections stained with osmic acid the fatty granules are found to be distributed irregularly within the cells, being largest and most numerous in the central parts around the nucleus; but even in advanced cases the whole of the muscle fibrils are never destroyed. It does not affect the size of the cells, but may be present in hypertrophied or atrophied cells.

Examination with a low power distinguishes two forms. In one (thrush breast) the distribution of the fatty cells is very irregular, little islets of more or less normal cells being surrounded by cells which are notably degenerate (Fig. 22). As a rule the transition is gradual, but sometimes a cell wholly free from fat may lie next one which is extensively disorganized. Sometimes almost every cell is fatty, but the patchy distribution is still apparent, islets of cells with comparatively little change lying within areas whose cells are greatly altered. In sections of the walls it is difficult to determine the relationship of the islets to the blood vessels,

but in sections of the musculi papillares, whose cells are arranged longitudinally, it can be seen that those *around* the arterioles are less affected than those farther away (para-arterial distribution). In the second form the fatty change is *diffuse* and almost equally distributed (Fig. 23).

The patchy degeneration is seen in its most exquisite form in the left ventricle in cases of anæmia, and is always best marked on the papillary muscles. It thus occurs in those

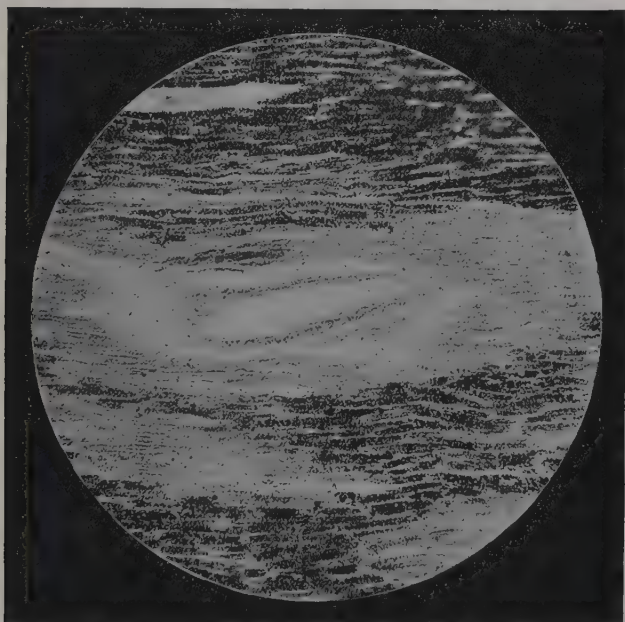


FIG. 22.—FATTY DEGENERATION: PARA-ARTERIAL DISTRIBUTION. ($\times 200$).
(From a case of anæmia.)

muscle cells that are farthest away from their nutrient arteries, and in those parts which are supplied by the longest branches of the coronary arteries. It is evidently due to a relatively insufficient supply of blood, arising partly from the blood condition, and partly from the anatomical arrangement of the vessels. In some cases the maximal change is situated elsewhere, the special site being determined by exceptional conditions. Thus in a case of aortic valvular disease the fat was maximal in the right ventricle, the orifice of the right

coronary artery being partially occluded by a patch of atheroma. In another case, that of a child who died as the result of burns, the right ventricle was more fatty than the left. The patient happened to be suffering at the time from bronchitis, the right ventricle was overtaxed, and the lesion, in consequence, appeared at the point of maximal functional strain.

The diffuse degeneration is of *toxic* origin. In local toxæmias, such as occur in abscess and pericarditis, a few fatty

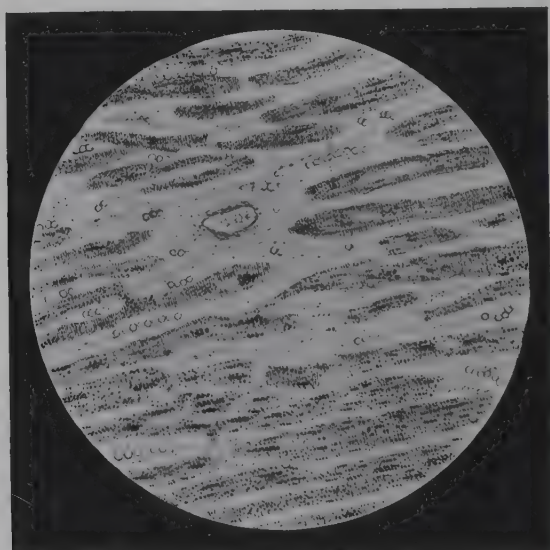


FIG. 23.—FATTY DEGENERATION : DIFFUSE FORM, OF TOXIC ORIGIN.
($\times 200$.)

cells can always be seen in the immediate vicinity of the lesion, and a widespread change can be readily produced in animals by the administration of chloroform or phosphorus. Clinically it is particularly associated with severe anæmias, emphysema, chronic renal disease and alcoholism. In the ordinary work of a laboratory it is difficult to secure "pure" cases of alcoholism, as many causes may have co-operated in producing death. In one of our cases, however, the association seemed definite. A man who had been drinking heavily for

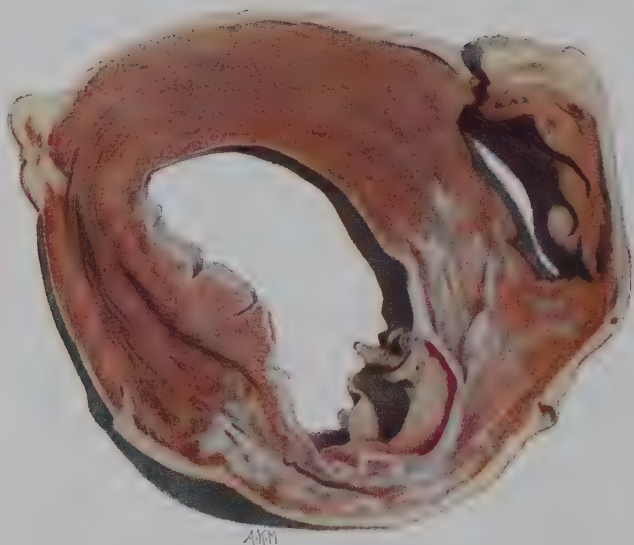


FIG. 24.—FIBROSIS OF THE ANTERIOR WALL OF THE LEFT VENTRICLE
(WESTERN INFIRMARY MUSEUM).

a fortnight was crushed between a cart and a wall, and died within a few hours from his injuries. His heart proved to be the site of an extensive and diffuse fatty degeneration.

A diffuse degeneration is never absolutely uniform in its distribution. The causes of death are often multiple. In pneumonia, for example, the maximum mortality occurs in alcoholics and in persons with degenerate organs. An acute infection is often the terminal event in a case of chronic renal disease with degenerate and narrowed coronary arteries. It is difficult in these cases to estimate the relative value of the different factors, the anæmia, toxæmia, high blood pressure, which together cause death, and to appreciate their precise effects upon the different parts of the heart. But given a blood poison it will act most rapidly on those parts of the heart which are working nearest to their maximum capacity.

The Fibroses of the Heart.—The connective tissue of the heart is not infrequently increased in amount. The fibrosis is never uniformly distributed, but is scattered irregularly in little islets or bands throughout the muscle. There may be only a single patch, or more frequently there are several, and they may be numerous. They may be of any size or shape; sometimes mere pinpoints, sometimes blocks an inch or more square; rounded or oval or star-shaped, or sometimes a long ribbon. Their consistence varies with their age, recent nodules being soft and vascular, the cut surface flush with the surrounding muscle; whereas the older patches are firm and depressed, the muscle standing out prominently around them. The wall may be notably thinned, and only measure a quarter of its normal thickness (Fig. 24).

Fibroid patches may occur anywhere, but are most common in the left ventricle (Fig. 25). The muscoli papillares are most often affected, and then, in order, the apical third of the left ventricle, the lower part of the septum, and the posterior wall of the left ventricle about the junction of its upper and middle third. The patches are generally situated deeply, and do not always extend to the surface, but in extreme cases the overlying endocardium is thickened and opaque, while the visceral pericardium may be similarly affected and is sometimes adherent to its outer layer.

Fibrosis is most frequently the result of obstruction of the coronary arteries. The anastomosis between the nutrient arteries is seldom sufficiently free to prevent the death of the area supplied by an artery if this becomes suddenly and completely blocked. The main branches anastomose with each other. *Sudden* and complete closure of a main branch produces an infarct, though the lesion is smaller

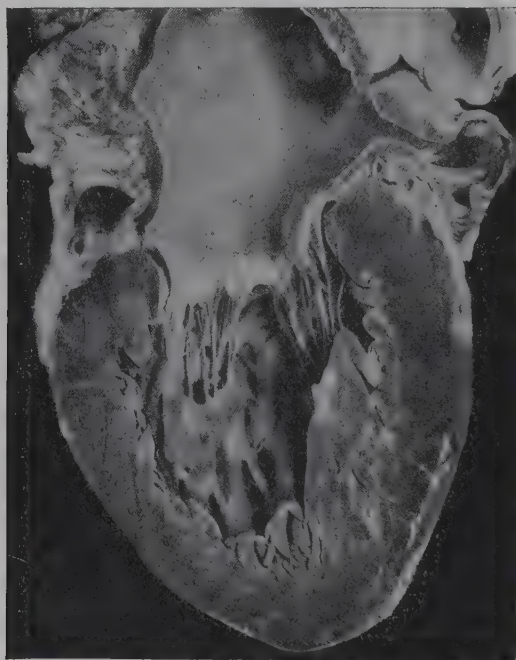


FIG. 25.—FIBROSIS OF THE LEFT VENTRICLE.

than the area anatomically supplied. But if the closure is *gradual*, compensatory enlargement of the other vessel may prevent the occurrence of any lesion. Several examples have been recorded, and we have seen at least five specimens. In one (Fig. 26), the descending branch of the left artery was completely obliterated about half-way down. The descending branch of the right artery was large, and could be traced round the apex upwards for an inch or

more in the anterior interventricular sulcus. In another case (Fig. 27) the orifice of the right artery was completely blocked by a patch of atheroma upon the aortic wall. The artery was greatly atrophied. The left artery was much enlarged, and the transverse branch gave origin to the posterior interventricular artery. There was no gross lesion in the muscle, though microscopic examination showed a few small patches of fibrosis, and, as the patient was able to perform all his duties until ten days before his death, the anastomosis must have been fairly efficient. Similar cases have been recorded; and a coronary artery has been ligatured on more than one occasion without evil effects.

The cardiac muscle may perhaps be supplied with blood in other ways. The foramina Thebesii are present in all the chambers of the heart, though they are most numerous upon the right side. They are generally considered to be venous in function, conveying blood from the muscle, but the evidence is inconclusive, and they may perform the function of

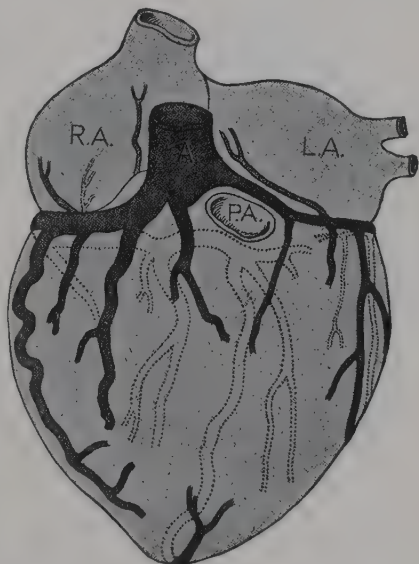


FIG. 26.—DIAGRAM SHOWING THE COMPENSATORY DEVELOPMENTS OF THE CORONARY CIRCULATION IN A CASE WHERE THE DESCENDING BRANCH OF THE LEFT CORONARY ARTERY WAS OCCLUDED.

arteries. The hearts of dogs, for instance, can be kept alive for several hours after removal, though the coronary arteries are ligatured, if a suitable fluid is allowed to pass into the cardiac cavities. The endocardial blood, too, can afford nourishment to the muscle, for, as Muir pointed out, in cases of healed infarct and in dystrophic fibrosis a thin layer of muscle always persists along the endocardial surface.

The coronary arteries are peculiarly liable to pathological

processes, and some change is almost constantly present after middle life. The orifice may be obstructed by disease of the aorta, or the main vessels may show patchy atheroma or diffuse fibrosis. Such changes can readily be seen upon naked-eye examination, but the condition of the larger vessels is no criterion of that of the smaller, whose walls may be seriously damaged without the larger vessels being affected. As a rule both sets are involved, but the degree of the damage is not necessarily comparable.

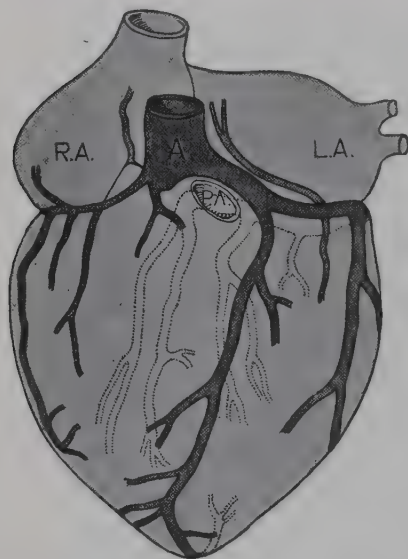


FIG. 27.—DIAGRAM SHOWING THE COMPENSATORY DEVELOPMENTS OF THE CORONARY CIRCULATION IN A CASE WHERE THE RIGHT CORONARY ARTERY WAS OCCLUDED AT ITS ORIGIN.

The muscle at the apex, that next to the endocardium, and the papillary muscles are supplied by the longest branches of the coronary arteries, and show most often and in greatest degree any lesions which are due to interference with the blood supply. Under normal conditions the work performed by the left ventricle is much greater than that done by the right, but in many diseases (emphysema, mitral stenosis, etc.) the right ventricle is overworked, and it is often, in consequence, hypertrophied.

Given a deterioration of the blood supply, whether in quantity or quality, the effect upon the muscle will be most manifest and will appear first where the blood supply is least ample, and where the greatest functional activity obtains. These, it will be found, are the cardinal factors in determining the site of almost all the focal lesions which occur in the myocardium.

Infarct. Myomalacia cordis.—The nutrient arteries may be occluded by emboli or thrombi, the latter being the more

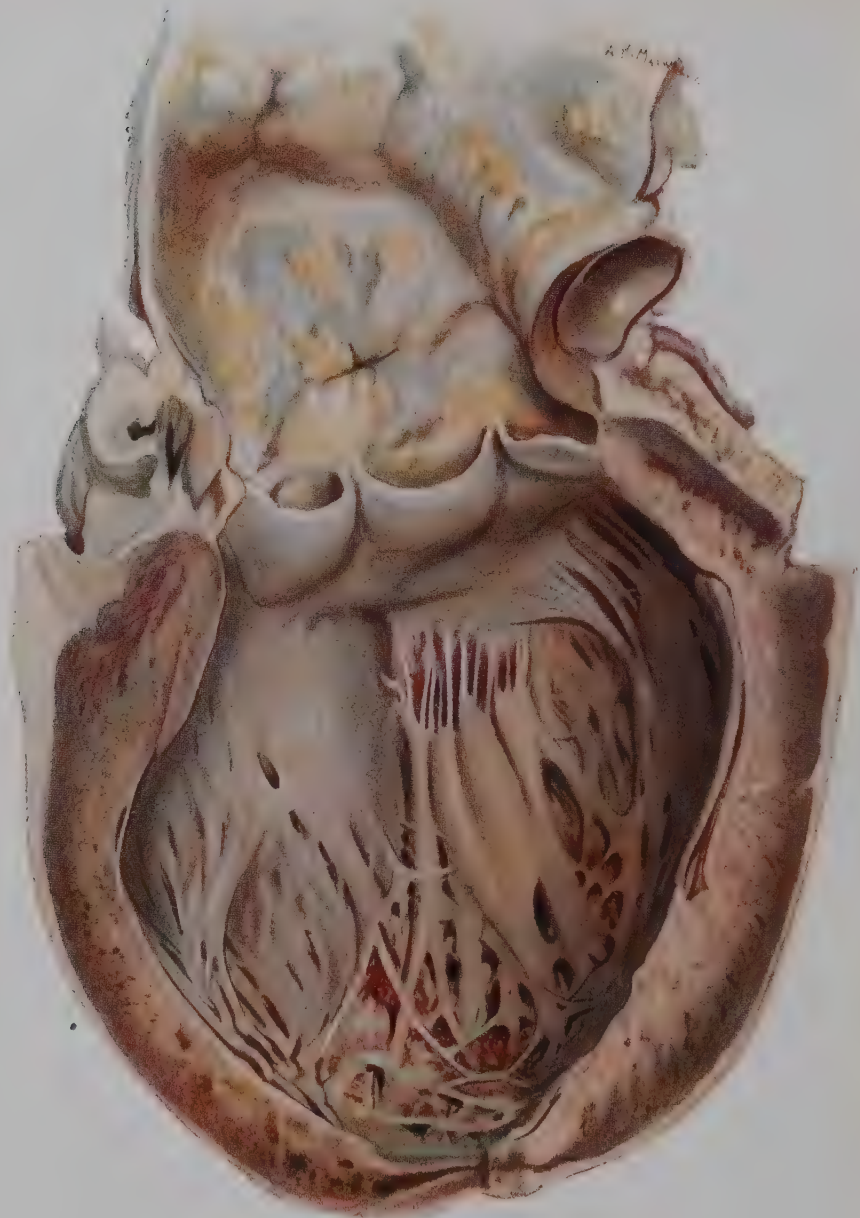


FIG. 29.—MARKED NARROWING OF THE ORIFICES OF THE CORONARY ARTERIES FROM SYPHILITIC DISEASE OF THE AORTA. INFARCT, WITH FATTY DEGENERATION OF THE WALLS OF THE LEFT VENTRICLE (GLASGOW ROYAL INFIRMARY MUSEUM).

common. The tissues which are involved necrose, and if the patient survives are gradually absorbed and replaced by connective tissue. Large infarcts are generally due to the sudden occlusion of a main artery, the abruptness of the closure preventing the successful development of the

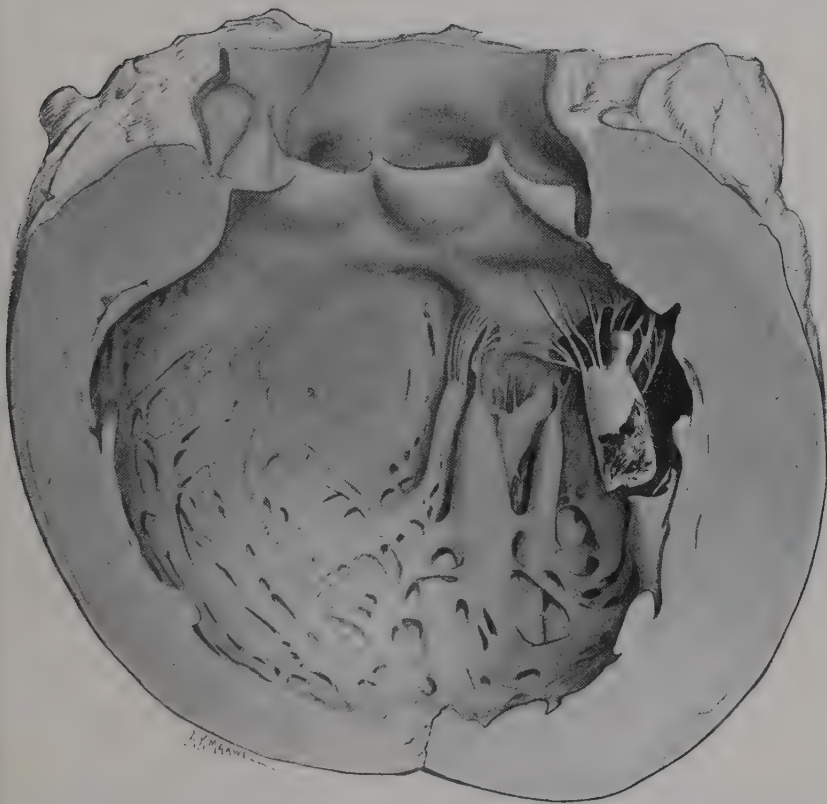


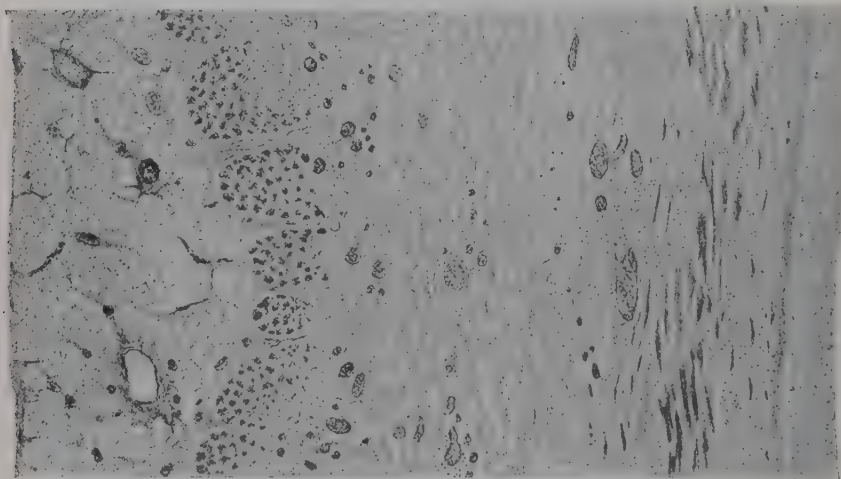
FIG. 28.—INFARCT OF A PAPILLARY MUSCLE, WITH RUPTURE, FROM DISEASE OF THE CORONARY ARTERIES (GLASGOW ROYAL INFIRMARY MUSEUM).

anastomotic connections. They occur near the peripheral distribution of the main vessels, near the apex between the extremities of the descending branches, and on the posterior wall of the left ventricle between the extremities of the transverse branches. The whole of the muscle fibres never disappear in these cases, a few cells always persisting at the

margins, kept alive by the endocardial blood and the sub-pericardial superficial anastomosis (Fig. 30).

Infarcts vary in their appearance. They may be dull yellow from fatty changes, or uniformly red or mottled from extravasation of blood. Sometimes they are white, in which case they are usually fairly firm in consistence; but they may be soft from hæmorrhagic or autolytic processes.

Para-arterial (Dystrophic) Fibrosis.—Partial occlusion of the arteries, or complete obstruction of gradual onset, leads



Pericardium

Endocardium

FIG. 30.—DYSTROPHIC FIBROSIS OF WALL OF LEFT VENTRICLE. ($\times 20$.)

Some muscle cells still persist along the pericardial and the endocardial margins.

to somewhat similar results, the muscle cells involved suffering in their nutrition, atrophying, and being replaced by connective tissue. The process is well seen in the papillary muscles.

Microscopic sections show that the distribution of the fibrosis is exactly similar to that of the fat in the para-arterial form of fatty degeneration, little islets of more or less normal muscle being embedded in diffuse patches of fibrous tissue. In the centre of the muscular islet an arteriole is visible, surrounded by muscle cells that are almost normal, while those

at the periphery show every grade of ischæmic atrophy (Fig. 31). The connective tissue in the centre of the fibrotic area is sparsely nucleated and often hyaline, but around the muscle it may be thickly nucleated and vascular.

In both infarct and para-arterial fibrosis the process is clearly dystrophic, the muscular degeneration being primary and the fibrosis secondary. The ultimate result, too, is similar, and in old-standing cases the precise mechanism of the process cannot be distinguished. Their usual cause is

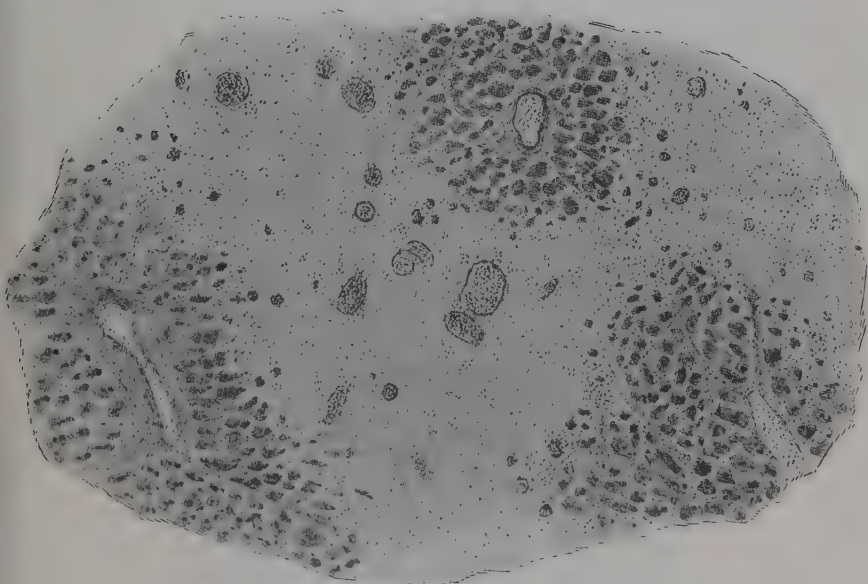


FIG. 31.—PARA-ARTERIAL (DYSTROPHIC) FIBROSIS. ($\times 50$.)

endarteritis of the coronary arteries, but they may follow occlusion of the orifice by a patch of atheroma in the aorta, as in the case reported by Gibson and Muir¹ and in one of our own cases. But coronary disease does not necessarily entail fibrosis. This only ensues when the lumen of the arteries is narrowed or occluded. The main vessels are often larger than normal when their walls are affected, though when the smaller vessels are involved the lumen is generally narrowed.

¹ Gibson, G. A., and Muir, R., *Edin. Hosp. Reports*, 1894, ii., 283.

Peri-arterial Fibrosis.—Peri-arterial fibrosis must be sharply differentiated from the para-arterial form. The latter is dystrophic. The former is inflammatory in nature.

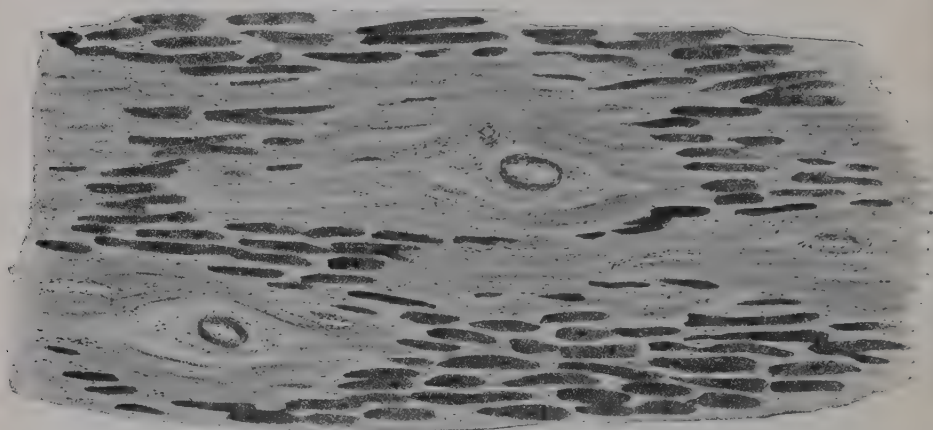


FIG. 32.—PERI-ARTERIAL FIBROSIS. ($\times 50$.)

In peri-arterial fibrosis the new-formed connective tissue is situated immediately around the arteries (Fig. 32), which

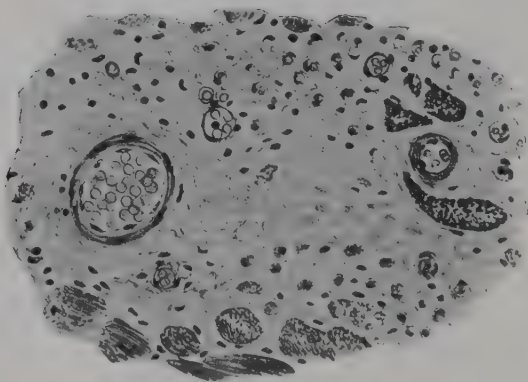


FIG. 33.—NEW-FORMED CONNECTIVE TISSUE IN THE VICINITY OF AN ARTERIOLE. ($\times 500$.)

(From a case of malignant endocarditis.)

are imbedded in an oval or star-shaped mass of variable size. In the smaller patches the muscle may not be implicated, but

in the larger ones the strands pass in between the muscle cells and separate them from each other (Figs. 33, 34). In early cases (Fig. 33) the connective tissue is cellular and vascular, but in older cases (Fig. 34) is often hyaline and sparsely nucleated.

Inflammatory lesions are not infrequently present in the walls of the heart. Large *abscesses* are unusual, for death occurs early in pyæmic infections which involve the heart, but microscopic ones are not uncommon. They may be found as a direct infection from an infected valve, a little

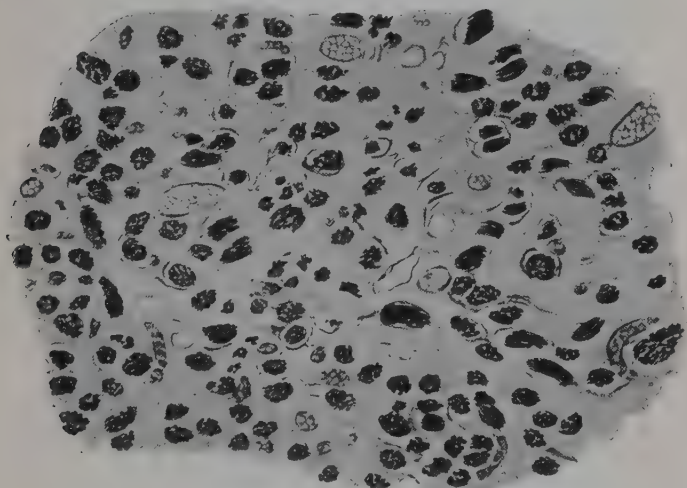


FIG. 34.—PERIFASCICULAR FIBROSIS. ($\times 200$.)

distance away under a patch of mural endocarditis, or disseminated widely throughout the muscle by a blood infection. In the majority of these cases death ensues during the period of infection; but a small number recover, and the scar beneath a thickened patch on the endocardium reveals the nature of the process.

In some cases of chronic mitral disease the inflammatory process is not limited to the valves, the cusps, the chordæ and the papillary muscles being all welded together firmly into a hard fibroid mass, the typical funnel-shaped mitral stenosis (Figs. 184, 187). The fibrous tissue in these cases is rarely confined to the areas mentioned, and extends from

the base of the valves into the adjacent muscle. A patchy fibrosis often co-exists.

It has been known for long that the rheumatic infection has a special predilection for the cardiac valves, and is, indeed, the most common cause of chronic cardiac disease. It was also recognized to be a frequent cause of the subcutaneous nodules which occur in childhood. But it was only in 1899 that Poynton¹ demonstrated the presence in the cardiac

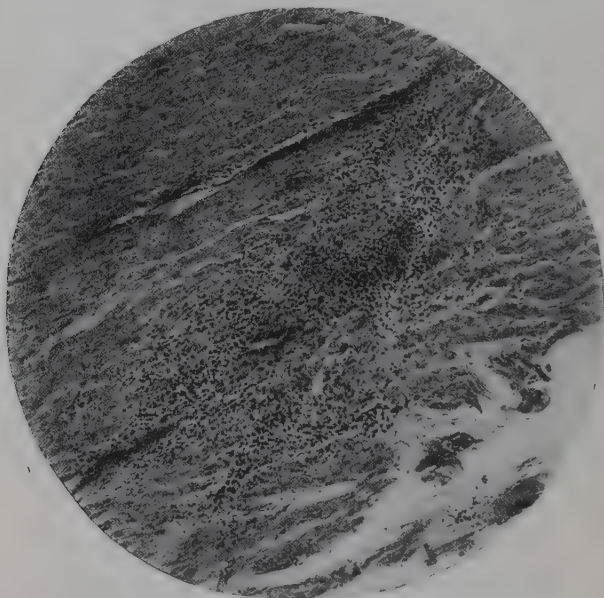


FIG. 35.—ROUND-CELLED INFILTRATION OF THE MYOCARDIUM: FOCAL FORM. ($\times 90$.) (A. M. K.)

muscle, in acute rheumatism, of *inflammatory nodules* which are exactly comparable to those found beneath the skin.

These nodules are microscopic in size, and occur most frequently in the left ventricle, generally in the vicinity of an arteriole. The essential lesion is a round-celled infiltration of the interstitial tissue. The cells may be few in number and the muscle apparently normal; or numerous and infiltrating the muscle, which is then degenerate. The vessel wall may be involved, and the lumen narrowed or even blocked

¹ Poynton, F. J., *Lancet*, 1899, ii., 1163.

by thrombi. In most cases the foci are discrete and widely separated from each other (Fig. 35), but in others the infiltration is diffuse (Fig. 37). The cells are mainly lymphocytes, but a few larger cells with a fair amount of protoplasm, evidently of connective-tissue origin, are often present, and occasionally a few multinucleated giant cells (Fig. 36). Some polymorphs, too, may be found. These infiltrations are most

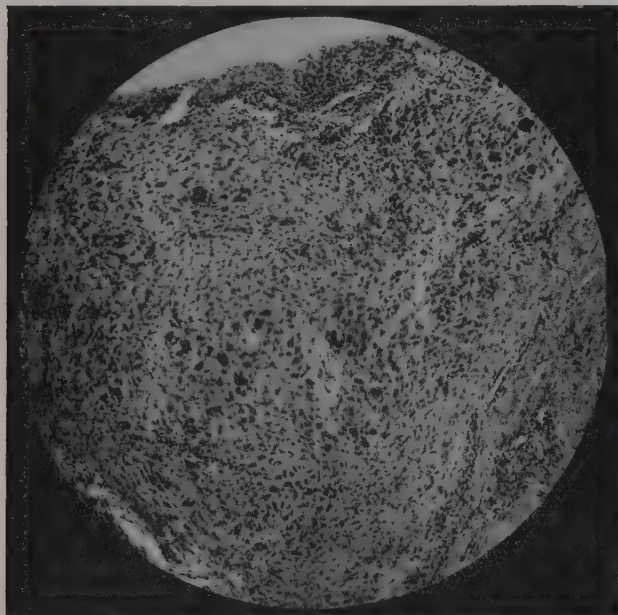


FIG. 36.—SUBMILIARY "RHEUMATIC" NODULE AT THE BASE OF THE TRICUSPID VALVE, SHOWING GIANT CELLS. ($\times 90$.) (A. M. K.)

common at the bases of the inflamed valves, but are also widely disseminated throughout the heart. Bacteria have not hitherto been found in them. The smaller nodules may perhaps be completely absorbed, but the larger lesions persist.

These nodules are not confined to cases of acute rheumatism, but occur in most of the acute infections—pneumonia, pleurisy, smallpox, blackwater fever, enteric fever, sepsis, diphtheria, scarlatina, chorea—and leukæmia. The individual lesion is minute and of comparatively little importance unless it

happens to involve the primitive tissues, the bundle or node, but their number may be considerable and consequently interfere with the proper working of the heart both in the acute and in the chronic stages. The heart, it must be remembered, cannot be immobilized like a broken leg, and its movements produce a "callus" quite out of proportion to the size of the original lesion.

The Mixed Form of Fibrosis.—Para-arterial and peri-arterial fibrosis may occur as individual lesions, but in perhaps

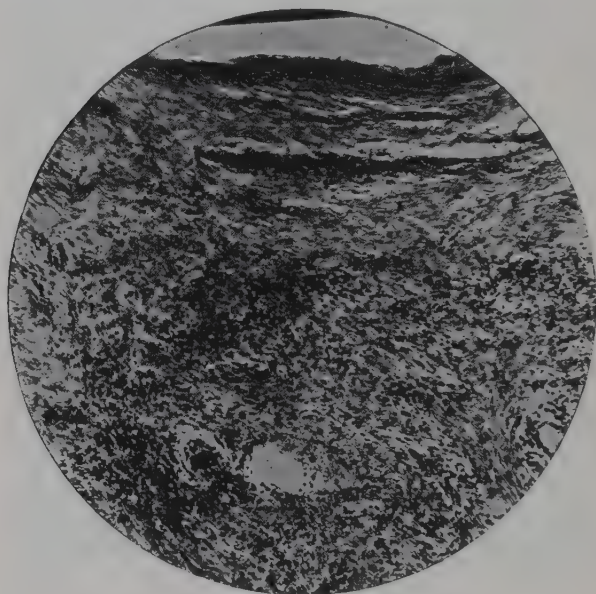


FIG. 37.—ROUND-CELLED INFILTRATION OF THE MYOCARDIUM: DIFFUSE FORM. ($\times 90$.) (A. M. K.)

the majority of cases occur together, though the amount of each varies in different specimens. This is the necessary result of the arterial cause. Endarteritis or adventitial lesions may be the sole arterial change in the early stage, but in advanced arterial disease both intimal and adventitial lesions co-exist, and both varieties of fibrosis occur.

The special localization of fibrosis is due to the frequency with which it is dependent upon disease of the coronary

arteries. Huchard pointed out that the left coronary artery, its descending branch in particular, is much more frequently abnormal than the right. The work which it has to perform is greater, extra strain is more frequently imposed upon it, and it divides rapidly into many branches. It is, too, less well protected than the right during the cardiac contractions. The arteries of the muscoli papillares are the longest branches of the system, with a course which is peculiarly tortuous,

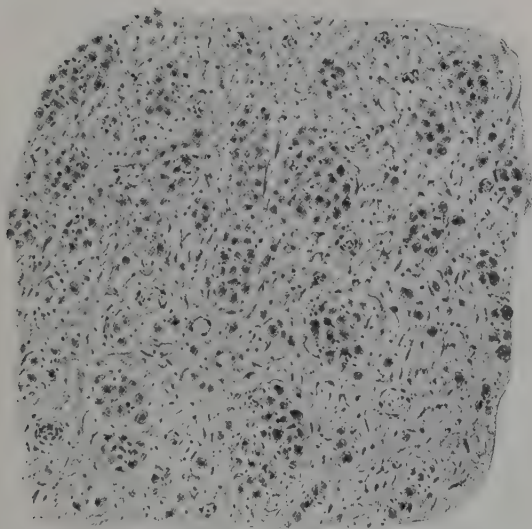


FIG. 38.—FIBROSIS OF THE MYOCARDIUM. ($\times 50$.)
(From a case of syphilis during the secondary stage.)

and therefore likely to suffer in disadvantageous circumstances.

The frequency of mitral endocarditis, too, with involvement of the neighbouring muscle in the inflammation, is another factor in determining the particular site of fibrosis.

The other causes of fibrosis are of comparatively little importance. *Hæmorrhages* are not infrequently found *post-mortem*. They are generally sub-pericardial, but also occur beneath the endocardium and in the muscle itself. They are usually small and cause little damage to the muscle, but they may be large, in which case the muscle in the vicinity is

degenerate. In such cases death as a rule occurs early, but if recovery ensues scar formation may conceivably result.



FIG. 39.—GUMMA OF HEART.

(From a water-colour drawing by Dr. Alex Macphail, Western Infirmary Museum.)

Tumours and tubercular masses are rarely present in the walls of the heart, and are usually secondary. *Hydatid cysts* have been occasionally found. *Wounds* of the heart, and

even *trauma* without penetration (Pleasants), may also lead to scar formation.

Syphilitic lesions may be present. The most important is syphilitic endarteritis, with a dystrophic fibrosis. Gummata (Figs. 39, 40) occur and may be responsible for some myocardial scars. A diffuse peri-arterial fibrosis (Fig. 38) is also recognized as occurring during the secondary stage of the acquired disease, and in congenital syphilis.

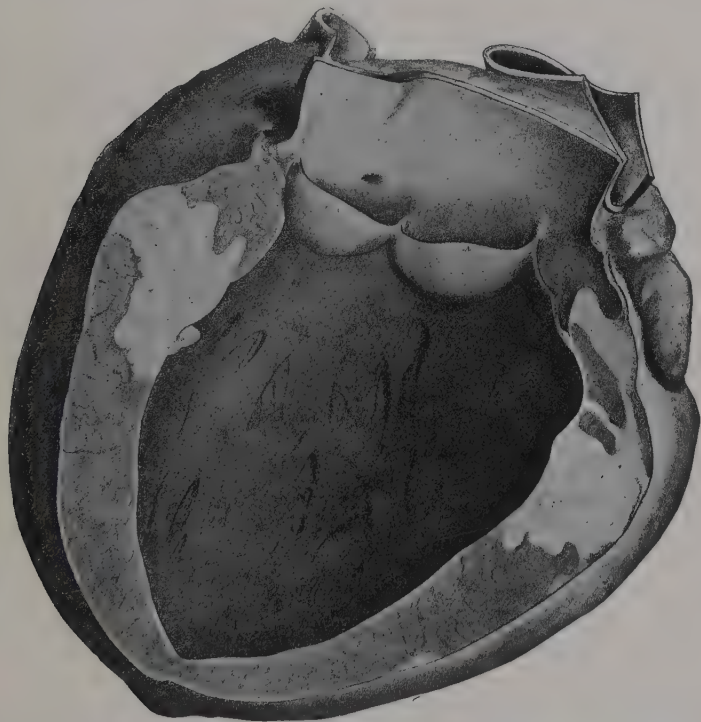


FIG. 40.—GUMMA OF HEART (GLASGOW ROYAL INFIRMARY MUSEUM).

Fibrosis of the heart may thus be due to several causes. In the great majority of cases, and in all gross lesions, it is the result of ischæmic destruction of the muscle following obstruction of the coronary arteries, which is frequently of syphilitic origin. In other cases it is an inflammatory affection. These two forms often co-exist. It may be due to mural endocarditis, but the lesion is never large. It

is rarely, if ever, due to pericarditis. Gummata and trauma may occasionally be the cause.

Chronic valvular disease seems always to be associated with some degree of fibrosis which is most extreme in the vicinity of the affected valve, though it occurs also in lesser degree throughout the heart.

Hypertrophied hearts are not necessarily fibroid, though this frequently results from associated arterial disease.

Aneurysm of the Heart is comparatively rare. It may be situated anywhere, but is most common in the lower parts of the left ventricle. The right ventricle is seldom implicated, and the auricles still less frequently. It may be of any size, and is sometimes as large as an orange.

Aneurysm is always secondary to myocardial disease which so weakens the wall that it gives way before the intracardiac blood pressure. The myocardial lesions may be of various kinds. Acute mural endocarditis is an occasional cause, usually affecting the septum. Myomalacia and dystrophic fibrosis are more common, and generally affect the lower parts of the left ventricle. A traumatic scar, the result of a stab ten years before death, has been followed by aneurysm.

In the majority of cases the wall is fibroid and greatly thinned, being sometimes only a fourth of the normal thickness. The endocardium is thickened and opaque, and often lined by laminated clot. The visceral pericardium is thickened and frequently adherent. The bulk of the muscle has disappeared, only a few cells persisting along the inner and the outer margins.

Rupture of the Heart is most frequently due to myomalacia. If an infarct occurs and the patient survives, an inflammatory reaction takes place at the periphery of the affected area, with resulting weakening of the tissues. If it extends near the endocardium the blood soon forces an entrance and, tearing its way along the track of the softening with successive heart beats, ultimately reaches and perforates the pericardium. The path of the blood may thus be devious, and the inner and outer lacerations may not correspond unless the communication is large.

Rupture has also followed trauma, intramural abscess, and aneurysm.

Dissociation.—The cells of the heart, when examined microscopically, are often seen to be dissociated. Sometimes the cleavage takes place along their line of junction (segmentation), each cell lying separate from its neighbours. Sometimes it takes place through the cell itself (fragmentation). The change may be limited and discrete, or widespread.

It has been suggested that dissociation is merely an artefact occurring during section cutting. Appearances due to this cause are often met with locally. But when it is widespread it is probably vital in origin. It is, however, present in some degree in the majority of hearts examined. It has been found in hypertrophied and in atrophied hearts, in hearts which are degenerate and in hearts which are sound, and in particular in the hearts of individuals whose end has been sudden (trauma, hanging). Its presence, then, seems to be of little importance, and it is probably due, as von Recklinghausen suggested, to irregular and excessive contractions of the cardiac muscle during the death struggle. Dissociation is not the cause, but a result, of death.

CHAPTER IV

THE EXAMINATION OF THE HEART

POLYGRAPHIC CURVES

THE investigation of the special functions of the cardiac muscle, its power of producing rhythmic stimuli, its excitability and contractility, the variations in the rate of conduction of the stimuli from part to part, and the tonicity of the organ as a whole, has been pursued in the laboratory for many years past; but it has only been possible within the last fifteen years, by the application of laboratory methods in the wards, to investigate the different functions in individual patients. By graphic methods the relationship of the contractions of the various chambers can readily be demonstrated, and the advance in our knowledge of cardiac disease has been, in consequence, considerable. Much work has been done, and many problems have been solved, but many more await solution. Some of the current theories may be disproved in course of time, and others may be verified, but the graphic methods of investigation have added greatly to precision in diagnosis, prognosis, and treatment; and a proper conception of the processes which produce cardiac failure in the individual case can only be attained by appreciation of the character and the site of the special disturbance. We are now able, even without any instrumental aid, to recognize many of these disturbances and to differentiate them one from the other, but it is on the evidence presented by polygraphic curves and electrocardiograms that our knowledge is founded.

The Interpretation of Polygraphic Curves.—The causes producing the variations of pressure within the cavities of the heart are not yet accurately known, and the data available in clinical work, though numerous, are obscure. The

main points, however, can be ascertained by analysis of tracings taken simultaneously from the vessels of the neck and from the apex impulse or the arterial pulse. In clinical work the most convenient instrument for this purpose is Mackenzie's polygraph, which also records in fifths of a second the speed at which the instrument is running.

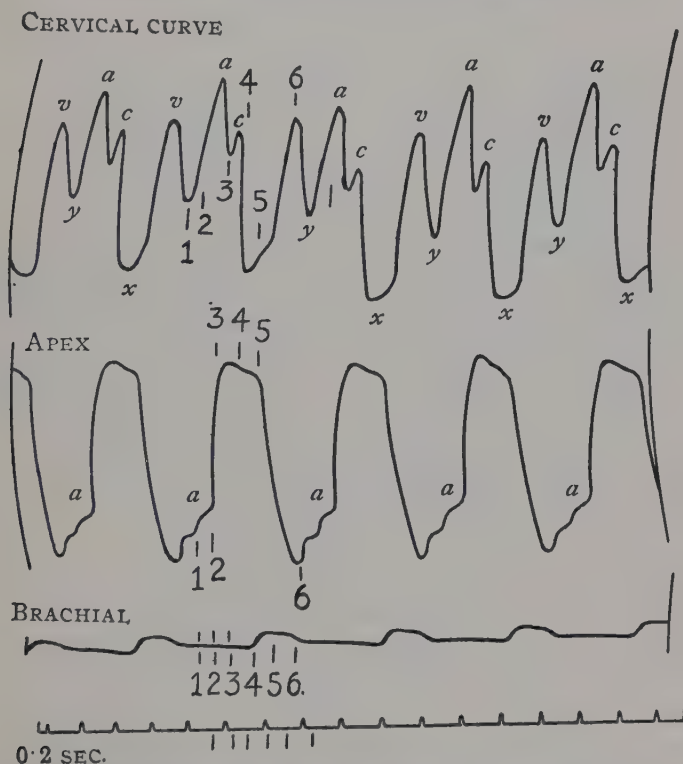


FIG. 41.—SIMULTANEOUS TRACINGS FROM THE JUGULO-CAROTID PULSATIONS, THE APEX-IMPULSE AND THE BRACHIAL ARTERY.

a, Auricular wave ; *c*, carotid wave ; *v*, ventricular wave ; 1, beginning of auricular wave ; 2, beginning of ventricular systole ; 3, beginning of carotid wave ; 4, beginning of brachial pulse wave ; 5, end of ventricular systole ; 6, opening of auriculo-ventricular valves.

In Fig. 41 the upper curve is from the right side of the neck, immediately above the clavicle, and just outside the sternomastoid muscle. The central curve is from the apex impulse ; while the lowest curve is from the brachial artery at the level

of the elbow. Below this the time is recorded in fifths of a second. The curves are not strictly comparable, because the upper, cervical, and the lowest, brachial, curves are records of changes in the volume (mainly) and the pressure within the vessels, whereas the central, apical, curve is the record of the varying relation of the apex impulse to the chest wall, and not of the intraventricular pressure.

THE APEX IMPULSE tracing shows, as a rule, one well-sustained wave and several smaller ones, the large wave representing ventricular systole (Fig. 41). Three phases of the latter can be recognized; the more or less abrupt line of ascent, beginning at the point marked by the numeral 2 in Fig. 41; the flat-topped plateau of the summit which may be broken up into several minor waves; and the abrupt fall to the base line. During the first phase, the presphygmic interval, the intraventricular pressure is rising until it becomes greater than the pressure within the aorta, when the semilunar valves open and the blood commences to flow out of the heart. The exact time of this occurrence is not indicated precisely upon the curve, and, indeed, the presphygmic interval varies in different cases from 0.02 to 0.085 second, from variations in the height of the aortic blood pressure at the end of diastole and in the relative strength of the ventricular systole.

During the period represented by the systolic plateau the output is maintained, and about its termination diastole commences, and the semilunar valves close. With relaxation of the ventricle the curve rapidly falls, and as soon as the pressure within the auricles exceeds that within the ventricles the auriculo-ventricular valves open, an occurrence which coincides fairly accurately with the lowest point upon the apex tracing, and is indicated in Fig. 41 by the numeral 6. Succeeding the fall there is often a well-marked wave, an instrumental fault due to the momentum of the recording lever, and best marked in cases where the antecedent fall is most abrupt.

As diastole proceeds, the ventricle again becomes distended with blood and the curve rises gradually. Towards the end of ventricular diastole the small wave *a* is seen, the result of the contraction of the left auricle. This wave is not invari-

ably present in the tracing, and may be absent although other evidence of auricular activity is undoubtedly present.

THE ARTERIAL TRACING.—The rise in the arterial curve is distinctly later than the commencement of the ventricular wave in the apex tracing, the delay being due to the presphygmic interval, and to the time occupied in the transmission of the pulse wave from the aorta to the brachial, or radial artery

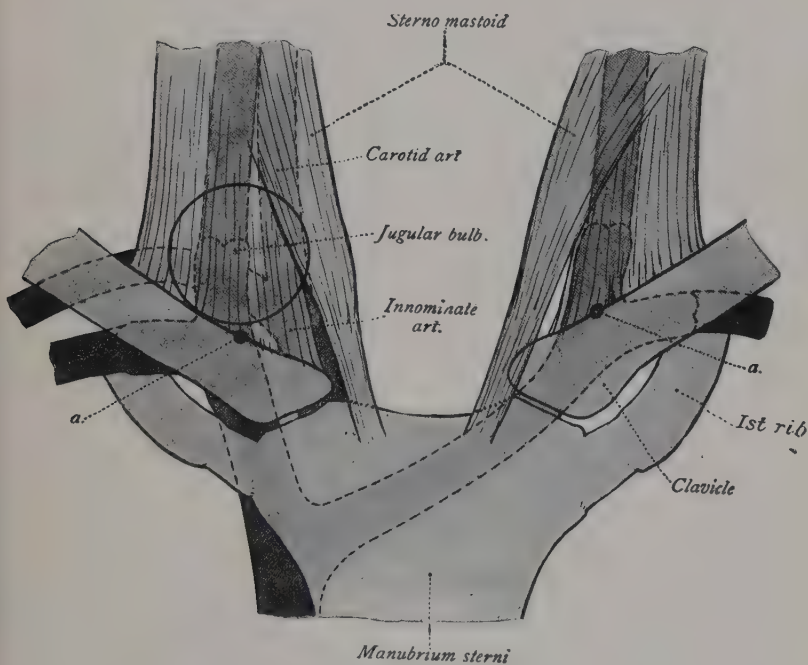


FIG. 42.—DIAGRAM SHOWING THE SITUATION OF THE CAROTID ARTERY AND THE JUGULAR VEIN. (A. KEITH.)

The circle shows the position of the receiver of the polygraph.

(Fig. 41), a period which necessarily varies according to the distance between the two points. The average period elapsing between the commencement of ventricular systole, on the apex tracing, and the appearance of the pulse wave at the level of the elbow is 0.13 second, but in Fig. 41 the period is 0.20 second.

THE CERVICAL, OR JUGULO-CAROTID CURVE.—This presents a variable number of waves which are due to various causes.

The receiver of the polygraph is placed immediately over the carotid artery and the jugular vein (Fig. 42), and the resultant tracing is the complex of the variations in volume and in pressure which are occurring in these vessels. With each beat of the heart there are three main waves, which are indicated in the upper curve of Figs. 41, 43, by the letters *a*, *c*, and *v*.

c Wave.—The first essential in the interpretation of the curve is the detection of the carotid wave *c* (3), which makes its appearance, as can be ascertained by measurements taken from the ordinates, shortly *after* the commencement of ventricular systole (2), as shown on the apex tracing, and shortly *before* the appearance of the pulse wave in the brachial or radial artery (4), and may be identified by reference to either.

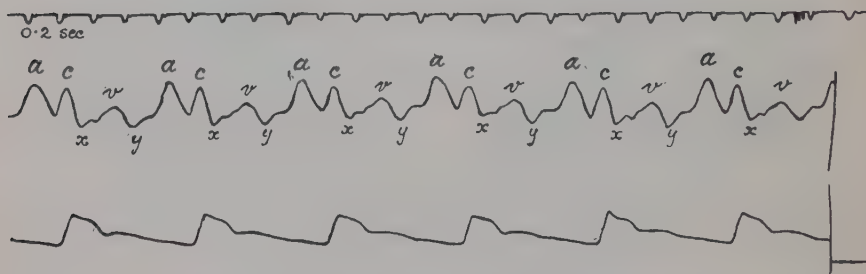


FIG. 43.—NORMAL RHYTHM, JUGULO-CAROTID AND BRACHIAL TRACINGS.

The carotid wave, *c*, usually begins about 0.06 second after the beginning of ventricular systole (*see* Fig. 41) as recorded in the apex tracing, and precedes the brachial pulse wave by 0.07 second. If there is any doubt as to the identity of the carotid wave in the curve, the receiver may be moved higher up the neck above the valves in the jugular vein, and a pure carotid tracing obtained for comparison.

There has been some discussion as to the precise significance of the *c* wave, for a wave has been recorded at practically the same time in tracings of intra-auricular pressure, as well as in the tracings from the jugular vein of animals, which has been dissected out free from all connection with the carotid artery. The wave may, therefore, be due to causes operating within the jugular bulb. The wave in question, when recorded experimentally, arises during the period of

ventricular systole, and is probably due to the ballooning upwards of the right auriculo-ventricular valve during that event. It is not invariably present in all tracings of intra-auricular pressure, and is usually small. In clinical work, however, the wave *c* is essentially arterial in origin. Firm pressure of the receiver in the neck produces a wave of typical arterial form, and, in the vast majority of cases, the *c* wave is due to the carotid pulse.

a Wave.—Immediately preceding *c* is the wave *a*. It occurs about 0.15 to 0.20 second before *c*, and synchronously with, or immediately before, the *a* wave on the apex tracing, and is the result of auricular contraction. There is little delay in its appearance, for there are no valves between the jugular bulb beneath the receiver and the auricle, and the distance is short. The variations in pressure within the auricle are thus rapidly transmitted to the jugular vein. The wave has been ascribed to regurgitation into the veins during auricular systole, and to stasis in the veins without regurgitation following the sudden rise of intra-auricular pressure during its systole. From the practical point the distinction is immaterial, and the wave must be considered as definite evidence of auricular contraction.

v Wave.—The wave *v*, following *c*, varies considerably in different cases both in the time of its occurrence and in its form. It is usually separated from *c* by the negative depression *x* (Figs. 41, 43), and may be single or double (*v*₁ and *v*₂). In the interpretation of the tracing it is important to note that the apex of *v*, or the hollow between its two peaks, coincides with the opening of the auriculo-ventricular valves. This event is marked 6 in the tracing (Fig. 41).

The early part of the wave occurs during ventricular systole, the notch between the two peaks being generally synchronous with the termination of the ventricular plateau (5) on the apex tracing. With the onset of ventricular systole the auriculo-ventricular valves are closed, the pressure in the ventricle being high and the pressure in the auricle at a minimum. During ventricular systole venous blood steadily enters the auricle from the venæ cavæ, and the intra-auricular pressure rises steadily. On the completion of ventricular systole the auriculo-ventricular valve opens, blood flows

from auricle to ventricle, the intra-auricular pressure falls, and the wave dies away.

The second wave, v_2 (Fig. 44), however, occurs after the ventricular systole has ended, and is therefore ventriculo-diastolic in time. During systole the apex of the heart

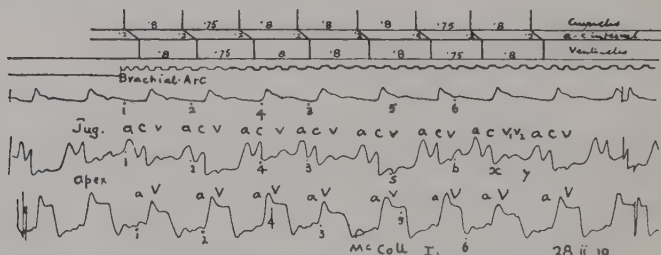


FIG. 44.—SIMULTANEOUS TRACINGS FROM THE BRACHIAL ARTERY, JUGULO-CAROTID PULSATIONS AND APEX-IMPULSE.

Above the tracings the time is recorded in tenths of a second.

is the only part which remains constant in position within the chest. During auricular systole the auriculo-ventricular junction is pulled upwards towards the great veins, and during ventricular systole pulled downwards towards the apex (Keith). This downward movement, expanding the auricles, is probably the chief cause of the negative depression x .

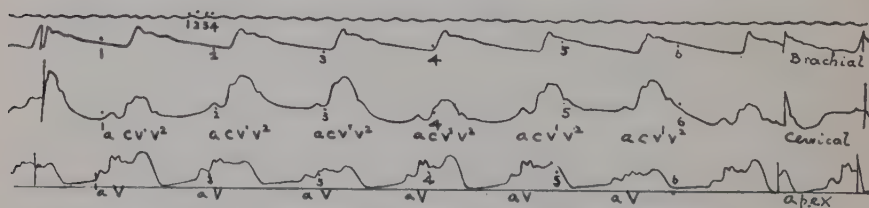


FIG. 45.—BRACHIAL, CERVICAL, AND APEX CURVES.

The negative depression, x , is absent from the cervical curve. The time-record is one-tenth of a second.

When ventricular systole ceases the pull upon the auriculo-ventricular junction ceases too, and it resumes its mean position. The rebound, when it is active, produces the positive wave, v_2 , in the jugular curve.

In certain cases v may occur earlier in the tracing, and may even merge in c , as in Fig. 45. This may be due to two causes.

v will appear the earlier the more rapid the diastolic filling of the auricle, which will occur whenever there is venous engorgement. It will also occur early if the auriculo-ventricular valve is incompetent and blood regurgitates into the auricle at the commencement of ventricular systole.

The depression x (Fig. 41) has already been ascribed, in part at any rate, to the negative pressure produced in the auricle by the displacement of the auriculo-ventricular junction. Another factor probably co-exists. During ventricular systole blood is forced out of the chest into the arteries, the intra-pleural pressure falls, and the intra-auricular pressure follows suit.

The depression y (Fig. 41), which follows v , is the result of ventricular diastole. As soon as the auriculo-ventricular valves open, blood flows from auricle to ventricle, and the auricular and jugular pressures fall until, with the lapse of time, both auricle and ventricle are again filled with the inflow of the venous blood, and, in consequence, the curve again rises.

There has been much difference of opinion as to the precise time of the closure of the semilunar valves, and the opening of the auriculo-ventricular valves.

It seems quite certain that the time of the opening and closure of the semilunar valves cannot be recognized upon the apex tracing. The valves open synchronously with the appearance of the *aortic* pulse, and close coincidently with the occurrence of the aortic dicrotic notch, but experimental curves show that these events have no constant relation to the apex tracing. The valves open during the line of ascent, and close before, at, or after the termination of the plateau. The transmission time, too, varies, and corresponding events in the carotid or brachial pulses cannot be related accurately to the same events in the aorta.

A similar conclusion must be arrived at with regard to the period when the auriculo-ventricular valves open. It seems certain that they close synchronously with the commencement of the upstroke of the systolic plateau, and that they open, after the ventricular systole has ceased, as soon as the relaxation of the ventricles has lowered the intra-ventricular pressure below that in the auricles. But the pressure within the

auricles, at the commencement of ventricular diastole, may vary even in the same case from varying degree of venous engorgement, varying length of ventricular systole, etc. The rate at which the intraventricular pressure falls is also inconstant. The cervical curve is a more accurate guide, and the apex of v , or the notch between v_1 and v_2 , may be considered as synchronizing with the opening of the auriculo-ventricular valves. In the brachial tracing this coincides approximately with the foot of the dicrotic notch.

The *a-c* interval in the cervical curve measures, as a rule, 0.15–0.20 second, and is used as an index of the time occupied by the transmission of the stimulus from the sinus node to the ventricles. If the *a-c* interval exceeds 0.20 second the con-

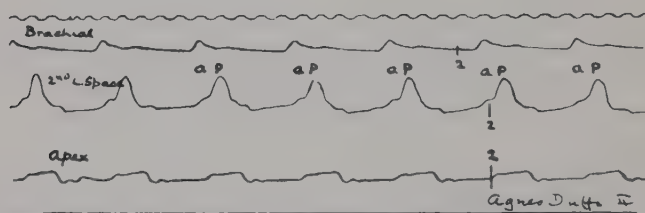


FIG. 46.—BRACHIAL AND APEX CURVES, AND TRACING (CENTRE) FROM SECOND LEFT INTERCOSTAL SPACE CLOSE TO THE STERNUM.

The wave *P* is evidently derived from the pulmonary artery, and succeeds the rise of the ventricular wave on the apex tracing. The interval between these events is the presphygmic interval.

duction of the stimulus is probably defective and unduly prolonged. Small variations from the average, however, may be produced by other causes.

During the *a-c* interval five events occur: (1) the contraction of the auricle; (2) the passage of the stimulus from the auricle to the ventricle; (3) the latent period, after the stimulus has reached the ventricle, before ventricular systole ensues; (4) the presphygmic interval; (5) the time occupied by the transmission of the pulse wave from the aorta to the carotid artery. The *a* wave is not an accurate index of the duration of auricular systole, for experimental evidence points to the continuance of auricular contraction after ventricular contraction has commenced. We have no knowledge of the duration of the latent period, but we know that the presphygmic

interval varies considerably (0.02–0.085 second) in experimental curves, and that the transmission time depends upon the distance traversed, the height of the aortic pressure at the end of ventricular systole, and the pressure reached during the presphygmic interval. Clinically the presphygmic interval and the transmission time cannot be estimated separately, but together (commencement of systole in the apex tracing to the appearance of the *c* wave in the cervical curve) they may vary from 0.03 to 0.11 second.

h or *b* Wave.—In slowly beating hearts a wave (*h* or *b*) may be seen in the cervical curve between each *v* and the succeeding *a* wave (Fig. 47). Its time relation to the preceding *c* wave is constant in the particular case (about 0.50 second), and the wave has been ascribed by A. G. Gibson¹ and Hirschfelder² to the snapping together of the cusps of the auriculo-

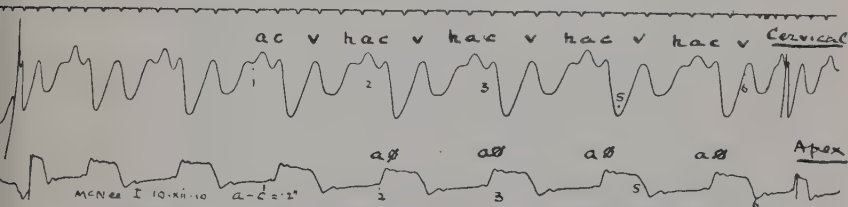


FIG. 47.—CERVICAL AND APEX CURVES, SHOWING THE WAVE *h* OR *b* IN THE CERVICAL CURVE.

ventricular valve at the end of ventricular filling in mid-diastole. It is at this period that the third sound of the heart is heard. The third sound is distinctly audible in some young persons, when the rate of the pulse is infrequent, and especially if they are lying upon their left side. In our experience the sound is not nearly so common as Thayer³ asserts.

In œsophageal tracings a similar wave may be present.

When the pulse rate quickens the *h* wave and the third sound disappear.

In determining the length of the *a-c* interval the possibility of *a* and *h* being mingled together, whereby the apparent

¹ Gibson, A. G., *Lancet*, 1907, ii., 1380.

² Hirschfelder, A. D., *Johns Hopkins Hospital Bull.*, 1907, xviii., 265.

³ Thayer, W. S., *Boston Med. and Surg. Journ.*, 1908, clviii., 713.

a-c interval becomes unduly prolonged, must be borne in mind. The fallacy can be detected by reference to the apex tracing, which will show that the *a* wave commences appreciably later than the cervical wave in question.

THE ELECTROCARDIOGRAPH

When any point of a muscle or other excitable tissue is stimulated, a change of electric potential is induced. The part which is stimulated and in activity becomes electro-negative to the parts at rest, and may be regarded as corresponding to the zinc plate of a galvanic cell; the remainder of the muscle, which is at rest, corresponds to the copper plate. If the active and passive portions of a muscle are connected by means of electrodes to a sensitive galvanometer, the changes in electric potential accompanying muscular activity—the action currents of the muscle—can be recorded by the instrument. Waller, in 1887, was the first to register the action currents of the human heart. He employed a capillary electrometer, and showed that they were transmitted throughout the body, and could be led off from moist skin surfaces. But as action currents are extremely feeble, and as the interpretation of capillary electrometer curves is attended with considerable difficulty, Einthoven employed the string galvanometer, and it is this instrument, with his modifications, which is now used extensively in physiological and clinical work.

The string galvanometer is based on the principle that a moving conductor (a fine fibre), suspended in a magnetic field at right angles to the lines of force, tends to be deflected to one side or other according to the direction of the current. The extent to which the fibre is deflected varies in direct proportion to the intensity of the current passing through it, and to the strength of the magnetic field, and inversely to the weight and tension of the fibre. In Einthoven's string galvanometer the currents are led through an extremely fine silvered glass or quartz fibre, which is stretched between the poles of a powerful electro-magnet. Accompanying each contraction of the heart there are changes of electric potential; the fibre oscillates to and fro, and its magnified shadow is

projected upon a moving photographic plate. The record thus obtained is termed an electrocardiogram.

The records should be taken while the patient is recumbent or reclining quietly in a chair, in order to avoid as far as possible all tremor of the somatic muscles, which distorts the electrocardiogram. The currents are led off from the two hands, or from one hand and one foot, immersed in porous jars filled with warm saline solution. Each jar lies within an earthenware pot containing zinc sulphate solution and an amalgamated zinc rod, which can be connected to the end of the fibre. The procedure is consequently not more irksome to the patient than that entailed in obtaining a graphic record by means of the polygraph. As compared with the polygraph, the string galvanometer has the great advantage of eliminating the fallacies dependent upon inertia, and upon the delay in the transmission of mechanical impulses. Its chief value, however, consists in the fact that by it alone do we obtain information regarding the essential nature of each heart-beat.

The form of the electrocardiogram varies according to the derivation that is employed. Following Einthoven's nomenclature, a lead from the right hand and left hand is termed "Derivation *I*," that from the right hand and left foot is "Derivation *II*," and that from the left hand and left foot is "Derivation *III*." When recording electrocardiograms it is customary to adopt Einthoven's standard, and to adjust the tension of the fibre so that, with a constant magnification of 450 diameters, its shadow is deflected 1 cm. when a difference of potential of 1 millivolt is introduced into the circuit containing the galvanometer and the patient. It is, therefore, possible to compare the amplitude of the individual deflexions in different records.

The Electrocardiographic Deflexions

A normal electrocardiogram, although varying in different individuals and according to the derivation employed, has certain constant features. Fig. 48 shows a normal electrocardiogram by Derivation *II*. Each upward deflexion, *P*, *R*, and *T*, indicates that either the base or right side of the heart is electro-negative, and therefore in predominant activity,

whereas the downward deflexions, Q and S (Fig. 62), signify negativity, namely preponderant activity of the apex or left side of the heart. P is the auricular deflexion; Q , R , S , and T are ventricular deflexions. In some instances T is succeeded by another upward deflexion, U .

THE AURICULAR DEFLEXION.—In a healthy heart the auricular deflexion, P , is a simple upward deflexion by all three derivations. It begins when the stimulus for contraction is emitted from the sinus node, and the summit of P coincides with the onset of the auricular contraction. In health the deflexion is usually larger when recorded by Derivation II (P_{II}), than by Derivation I (P_I), or by Derivation III (P_{III}); but in auricular flutter (*see* page 156), and in some other abnormal forms of auricular activity, P_{III} is larger than

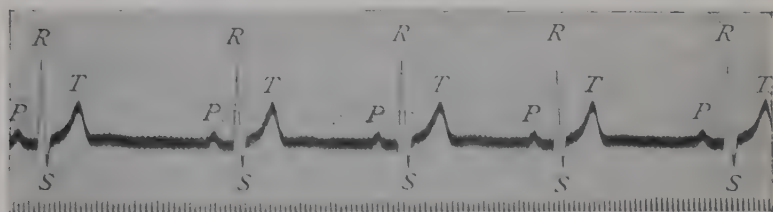


FIG. 48.—NORMAL ELECTROCARDIOGRAM BY DERIVATION II.

The time record is 28·57 per second, six divisions representing 0·21 second.

P_I or P_{II} . Although the amplitude of the electrocardiographic deflexions cannot be regarded as a definite measure of the electro-motive force of the heart, nevertheless P , and particularly P_{II} , is larger than normal in cases of auricular hypertrophy (Fig. 55). This is true especially in cases of mitral stenosis, in which disease the summit of the auricular deflexions may also be bifurcated (Fig. 54).

When the deflexions P_I or P_{II} are inverted it is generally supposed that the auricular contraction has started at some other site than the normal one in the sinus node. For example, the deflexion P is inverted in a nodal extrasystole (*see* Fig. 95), and P may be diphasic when the heart is retarded by vagal stimulation, or when the auricles are in flutter (*see* Chapter X). According to Rothberger and Winterberg, P becomes inverted when the left accelerator nerve is stimu-

0.10 second. During the period between S and T the main mass of ventricular muscle is in contraction; the action currents compensate one another, and therefore there is no predominance of activity either at base or apex or of one ventricle over the other. In health the terminal deflexion T is positive, its apex being upwards.

The Initial Ventricular Deflexions.—The constancy of the duration of the Q - R - S , or R - S , group of deflexions in healthy individuals points to the various parts of the ventricular muscle being activated in a constant manner. Undue prolongation of this group of deflexions beyond the normal 0.10 second indicates delay in the spread of the stimulus through the ventricular muscle; while irregular notching of the Q - R - S group indicates that the stimulus is not spreading in normal sequence to the various parts of the ventricles (see Intraventricular block, page 138).

The form of the ventricular complex depends in part upon the anatomical axis of the heart,¹ namely upon its inclination to the vertical or to the horizontal plane, and upon its change of position during inspiration and expiration. The form of the ventricular complex is, however, influenced more markedly by the electrical axis of the heart. We shall not describe the methods² whereby this axis can be calculated, because they are not essential in clinical work. For practical purposes, however, we have to remember that the form of the ventricular complex depends upon the ratio of activity of one ventricle to that of the other, in other words upon the balance of activity of the two ventricles. The amplitude of the deflexions R and S becomes altered when the normal balance of activity of the two ventricles is disturbed by preponderant activity of the right ventricle, as in mitral stenosis, or of the left ventricle, as in aortic incompetence and other diseases leading to preponderant hypertrophy of the left ventricle. In left ventricular preponderance R_I is larger than R_{III} , and S_{III} is larger than S_I (Figs. 50, 51 and 56 to 61). In right ventricular preponderance R_{III} is larger than R_I , and S_I is greater

¹ Waller, A. D., *Proc. Roy. Soc., Lond.*, 1913, cxxxvi., 507.

² Einthoven, W., Fahr, G., and de Wart, A., *Arch. f. d. ges. Physiol.*, 1913, cl., 275. Carter, E. P., Richter, C. P., and Greene, C. H., *Johns Hopkins Hosp. Bull.*, 1919, xxx., 162.

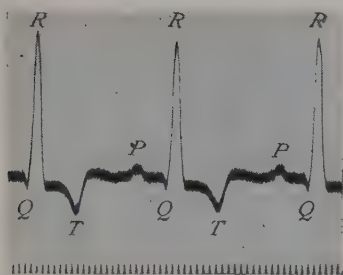


FIG. 50.

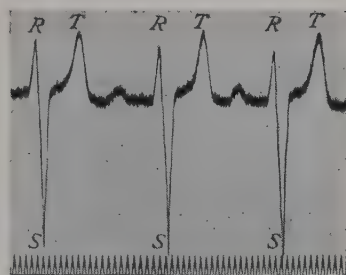


FIG. 51.

FIGS. 50 AND 51.—AORTIC INCOMPETENCE, MALE, AGED 19.

FIG. 50 IS BY DERIVATION I; FIG. 51 BY DERIVATION III.

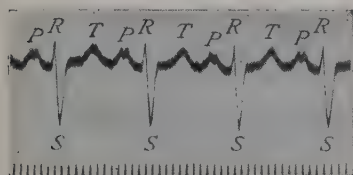


FIG. 52.

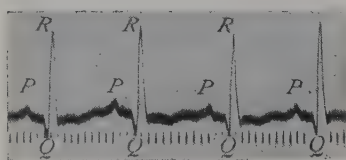


FIG. 53.

FIGS. 52 AND 53.—MITRAL STENOSIS, FEMALE, AGED 7.

FIG. 52 IS BY DERIVATION I; FIG. 53 BY DERIVATION III.

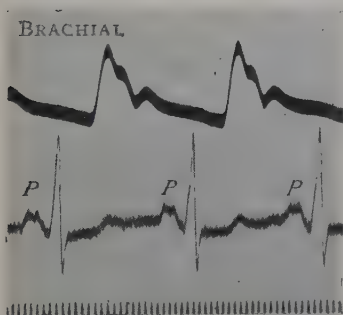


FIG. 54.

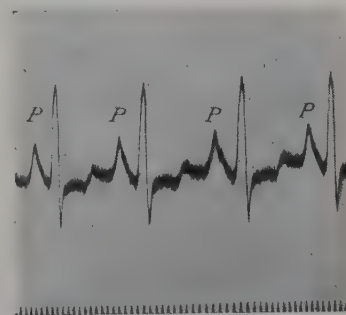


FIG. 55.

FIGS. 54 AND 55.—FROM CASES OF MITRAL STENOSIS, BY DERIVATION II.

These figures illustrate the electrocardiographic features of left and right ventricular preponderance.

(The above figures are reproduced $\times \frac{4.4}{5}$)

than S_{III} (Figs. 52, 53). Thus if, by Derivation I , R is large and S is small, whereas by Derivation III R is small and S is large, the record reveals left ventricular preponderance. On the other hand, if R_I is small and S_I is large, while R_{III} is large and S_{III} is small, then the record is one of right ventricular preponderance. In other words, the relative amplitude of R_I and S_I in right ventricular preponderance corresponds to that of R_{III} and S_{III} in left ventricular preponderance (Figs. 50 to 55).

The Terminal Ventricular Deflexion.—In health the terminal deflexion, T , is positive, its apex being directed upwards. This does not signify that the right ventricle remains in contraction longer than the left, nor does it indicate that when each contraction wave spreads through the ventricular muscle the last portion to contract is that near the aorta and pulmonary artery. From our clinical experience we would suggest that an upward T_I may be regarded as indicating predominant activity of the right ventricle over the left at the end of systole, whereas a downward T_I implies predominant activity of the left ventricle at the same period. For example, Fig. 50, from a case of aortic incompetence with preponderant activity of the left ventricle shows a negative T_I , whereas Fig. 52, from a case of mitral stenosis with right ventricular preponderance, shows a positive T_I . In right and in left ventricular preponderance the form of T_{III} may be the reverse of that shown by T_I . Thus, in left ventricular preponderance T_{III} may be positive and large (Fig. 51), whereas in right ventricular preponderance T_{III} may be flattened or negative (inverted) (Fig. 53).

The clinical significance of flattening of the deflexion T is not fully understood. T_I and T_{II} are normally positive and of considerable amplitude, T_{II} being larger than T_I or than T_{III} . When T_I or T_{II} are of low amplitude it does not necessarily follow that the ventricles are enfeebled. The small amplitude of the deflexion may be due to enfeeblement of the right ventricle or to greater activity of the left ventricle than in health. Similarly when the deflexion T becomes lowered after the administration of digitalis or of adrenalin this may be a temporary manifestation of increased activity of the left ventricle, and we know that this change of the

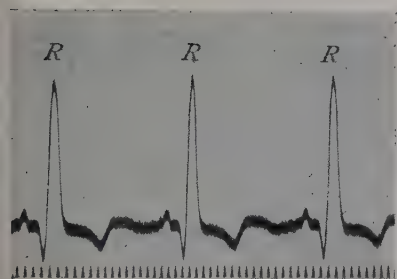


FIG. 56.

Electrocardiograms from a case of aortic incompetence by Derivation *I* (Fig. 56) and Derivation *III* (Fig. 57).

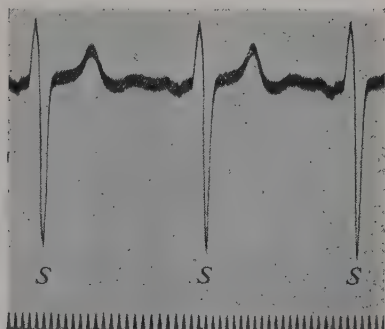


FIG. 57.

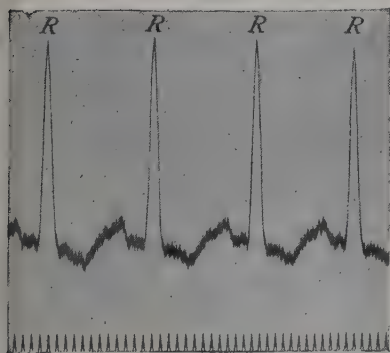


FIG. 58.

Electrocardiograms from another case of aortic incompetence by Derivation *I* (Fig. 58) and Derivation *III* (Fig. 59).

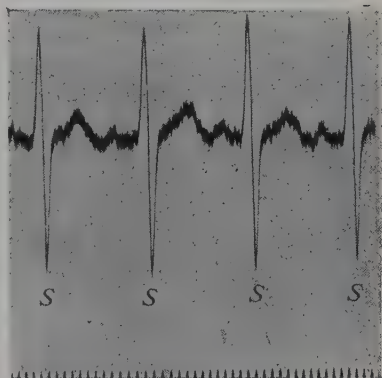


FIG. 59.

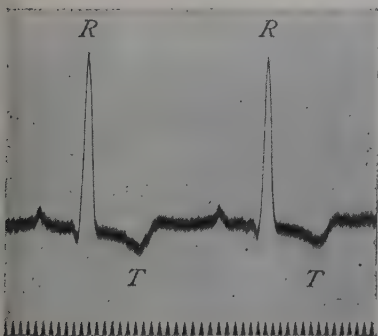


FIG. 60.

Electrocardiograms by Derivation *I* (Fig. 60) and Derivation *III* (Fig. 61) from a case of arterio-sclerosis without any valvular disease.

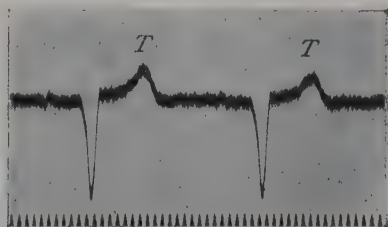


FIG. 61.

FIGS. 56 TO 61.—LEFT VENTRICULAR PREPONDERANCE.

deflexion T disappears while the heart is under the influence of atropine.

The R-T Period.—The duration of ventricular systole, as expressed by the R - T , or Q - T , period, is remarkably constant in health. It averages 0.32 second, the extremes in our cases being 0.28 and 0.36 second. In the vast majority of patients who present disease of the circulatory system the duration of the R - T period is likewise constant.

Prolongation of the R - T period is occasionally observed.

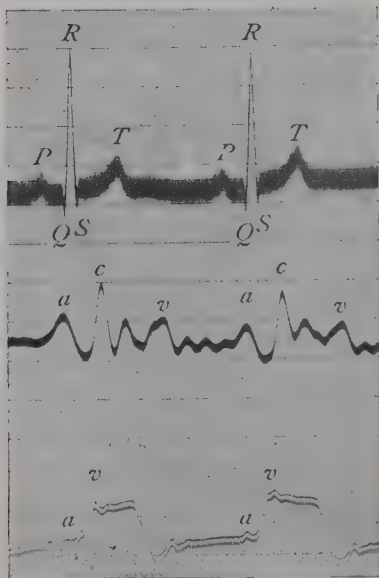


FIG. 62.—SIMULTANEOUS ELECTROCARDIOGRAM (DERIVATION II), JUGULO-CAROTID CURVE AND APEX IMPULSE. NORMAL HEART.

This is most frequent in complete auriculo-ventricular block; in twenty-three cases the average period was 0.49 second, the extremes being 0.24 and 0.66 second. Fig. 111, from a case of complete block together with block of the right branch of the bundle, shows an R - T period of 0.66 second. In nine cases of partial block the average R - T period was 0.423 second. Prolongation of this period may also be observed, though rarely, in patients with a normal, sinus, rhythm. For instance, in elderly patients the R - T period may be 0.44 second to 0.53 second. When the R - T , or Q - T , period is prolonged the

Q - R - S complex may be of normal length, or may be lengthened.

In ventricular extrasystoles, the average R - T period is 0.03 second longer than that of the sinus beats, but the extrasystolic R - T period may be as long as 0.5 or 0.6 second. The R - T period of auricular and of nodal extrasystoles, however, is often slightly shortened. Shortening of the R - T period, e.g., 0.24-0.28 second, may also be observed in auricular

fibrillation, flutter, and nodal rhythm when the ventricular rate is greatly accelerated.

*The Time Relations of the electrocardiographic deflexions to the waves of the normal jugulo-carotid pulsation, and to the apex impulse, are shown in Fig. 62. *P* is seen to precede *a*. The summit of *P*, which coincides with the beginning of auricular contraction, is about 0.04 second before the start of the *a* wave in the jugulo-carotid curve. The *Q-R-S* group of deflexions precedes the onset of ventricular systole, as determined by the apex impulse, for the latter begins synchronously with the lowest point of the deflexion *S*. Because of the presphygmic interval, and of the delay in the transmission of the pulse wave from the aortic valve to the carotid*

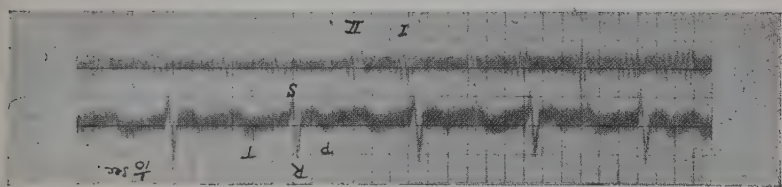


FIG. 63.—SIMULTANEOUS ELECTROCARDIOGRAM (DERIVATION II) AND PHONOCARDIOGRAM FROM BASE. NORMAL HEART. I=1ST SOUND; II=2ND SOUND. (H. L. WATSON-WEMYSS AND J. D. GUNN.)

artery, the carotid wave, *c*, is still later in relation to the initial ventricular deflexions and to the systolic upstroke on the apex tracing. The carotid wave begins almost synchronously with the commencement of the systolic plateau of the apex impulse, and follows the initial ventricular deflexion by 0.12 second. The normal duration of the *P-R* interval is 0.14 to 0.15 second.

NOTE.—Fig. 63 is inserted wrong way up.

¹ Watson-Wemyss, H. L., and Gunn, J. D., *Edin. Med. Journ.*, 1913, N.S., xi., 124.]

deflexion *R*, 0·02 to 0·03 second after the beginning of the first ventricular deflexion, and ends during the period *S-T*. The second cardiac sound occurs at the beginning of ventricular diastole, 0·05 second after the summit of *T* (Fig. 63), and precedes the opening of the auriculo-ventricular valves. The frequency of its vibrations is from 80 to 100 per second.

CHAPTER V

STIMULUS PRODUCTION

THE pulse of a healthy adult, at rest, beats fairly regularly about seventy times per minute. When he is in the erect posture the pulse-rate is increased by about six beats per minute, and it becomes more rapid on exertion or with emotion. In infancy and childhood, the rate is more frequent than in the adult, and often varies considerably, being more markedly accelerated by emotion or exercise and after the ingestion of food.

In health, under all these circumstances, stimuli formed rhythmically in the sinus node are transmitted by the normal paths to the auricles and to the ventricles, and these chambers contract in response to each stimulus. The sinus node is dominating the heart; in all other parts of the heart stimulus formation is in abeyance. The heart is therefore said to have a sinus rhythm, the essential feature of this being a sequence of contractions each of which starts at the normal site. The auricles contract before the ventricles; the pulse is regular except for phasic alterations in the length of diastole which coincide with the varying phases of respiration; a tracing from the jugular vein presents a normal curve with *a*, *c*, and *v* waves in succession; an electrocardiogram reveals an upward auricular deflexion before each normal ventricular complex (Fig. 48). In practice we are satisfied that the heart has a sinus rhythm if the pulse, at a rate of not less than 45 and, in adults, not more than 130, is either absolutely regular, or becomes faster during inspiration and slower during expiration.

All changes in the rate of a healthy heart are brought about by variations in the rate of stimulus production in the sinus node. This may be accelerated, retarded, or irregular,

and we therefore recognize (1) sinus tachycardia, (2) sinus bradycardia, and (3) sinus irregularity or sinus arrhythmia. In all these conditions the length of ventricular systole remains constant at about 0.3 second ; the auriculo-ventricular interval is also constant at about 0.14 second ; and the cardiac rate depends on the varying length of diastole, which may be shorter or longer than the normal average of 0.5 second.

SINUS TACHYCARDIA

In health, acceleration of the heart by physical exercise, by emotion, or by other factors always signifies an increased rate of stimulus production in the sinus node. The increase of rate is effected mainly by sympathetic stimulation, though the vagal control is probably lessened at the same time. That the acceleration depends on the nervous system is demonstrated by the increase in rate of the dog's heart from about 100 to 220 after section of the vagi, and by the acceleration of the human heart after injection of adrenalin which stimulates the sympathetic, or of atropine which paralyses the vagal nerve endings.

In disease, acceleration of the heart is usually a sinus tachycardia. The condition may be due primarily to *sympathetic hyper-excitability*, as in neurasthenia, in rapid irritable cardiac action ("D.A.H.," irritable heart of soldiers, neuro-circulatory asthenia), or in other functional nervous disorders. Many individuals of a nervous temperament have a markedly unstable pulse, which becomes much accelerated by trivial causes. The effect on a healthy heart of walking a hundred yards is to raise the pulse-rate from about 75 or 80 to 90 or 100 ; but within two minutes after the exercise has been completed the pulse falls to the pre-exertion rate. As a contrast to this we may cite the case of a neurasthenic man, aged thirty, of fair physique, complaining of palpitation and weakness. He had no organic disease of the heart ; the hands were tremulous, and the tendon reflexes were exaggerated. When recumbent, the pulse-rate was 120 ; when erect, 132 ; after walking one hundred yards the rate was 160, and two minutes later it was 108. Another well-recognized form of sinus tachycardia is that observed in young men while undergoing medical examination for life insur-

ance. In other cases the sympathetic hyper-excitability is due to more obvious causes, as in a soldier aged twenty-two who was ailing two years after he had been thrown to the ground by a shell-explosion. His pulse-rate at rest was 120, after bending and touching the toes six times it was 152, but the pre-exertion rate was regained in two minutes.

In other patients the sympathetic stimulation is probably secondary to organic or functional disturbance of the *endocrine organs*, as in exophthalmic goitre and other forms of hyperthyroidism, in which disorders the pulse-rate is often 100, 120, or more per minute, and rises to 150-200 under the influence of trivial emotional stress. Owing to the close inter-relation of the adrenals, of the thyroid, and of the sympathetic system, there may sometimes be doubt as to whether the primary etiological factor is glandular or nervous. But in "D.A.H." we very seldom find any clinical evidence of hyperthyroidism, and this is confirmed by laboratory tests which fail to show any excess in the percentage of blood sugar or any lowering of glucose tolerance. The response of these patients to injection of adrenalin may not be excessive, but in about 30 per cent. it reveals a sympathetic hyper-excitability.

In all these forms of sinus tachycardia, the pulse-rate at rest is more often about 90 than 70 or 80 per minute. On exertion the rate usually rises to 120 or 140, or may attain a still faster rate (160 to 180); but although the respirations may be much quickened, giving a veritable tachypnoea, there is no dyspnoea or distress on exertion. After exertion, the pulse-rate may decline rapidly, falling to the pre-exertion rate within two minutes, or the decline may be slow. The acceleration and the fall of rate are never abrupt; there is never a sudden transition, within the space of a single beat, from the slow to the fast, or from the fast to the slow, rate.

The cardiac rate is also accelerated in many forms of *poisoning*, as in those resulting from alcohol, tobacco, tea, or chloroform, and likewise in anæmia and in other debilitating diseases. In most *toxæmias* and *infective* diseases, especially if there be fever, the heart rate is almost invariably accelerated, the only exceptions being in the early stages of enteric, or when there is an increase of intracranial pressure, as in tuber-

culous meningitis. In pulmonary tuberculosis, a pulse-rate which is persistently above normal is often one of the early signs of the disease; the rate is more markedly quickened by exercise, and the decline to the pre-exertion rate is slower than in health. In pneumonia, in acute endocarditis and in other acute infections, the sinus rhythm is almost invariably preserved and accelerated, but in adults suffering from these diseases the pulse very rarely attains or exceeds a rate of 140 except as a terminal event. In the vast majority of patients convalescing from acute infections the rate is persistently above normal, and is easily quickened by trivial exertion; some weeks may elapse before the pulse-rate becomes altogether normal. In myocarditis and valvular

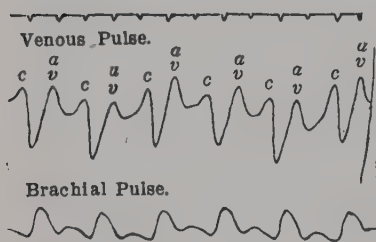


FIG. 64.

SINUS TACHYCARDIA. The rate of the heart is 135 per minute.

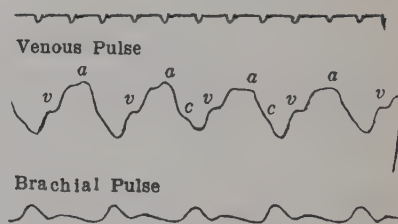


FIG. 65.

SINUS TACHYCARDIA. The rate of the heart is 120 per minute.

disease the sinus rhythm is usually maintained, even although the rate is accelerated to 100, 130, or more per minute, and although there is pronounced heart failure, as revealed by cyanosis, dyspnoea and dropsy. But in adults who are confined to bed on account of *heart failure*, a pulse-rate of 140, or more, always suggests the probability of an abnormal rhythm, and especially of auricular flutter or fibrillation or of nodal rhythm.

Jugular Tracings in sinus tachycardia may reveal the *a*, *c*, and *v* waves, each clearly shown. But when the rate approaches or exceeds 100, diastole is so greatly shortened that each *a* wave is superposed on, or mingled with, the antecedent *v* wave. For each cardiac contraction the tracing will therefore present only two waves, *c* and $\frac{a}{v}$ (Fig. 64).

There may be only one wave if the *c* elevation is not well represented.

Electrocardiograms present normal ventricular complexes. If the rate is much accelerated, the auricular deflexion, *P*, begins before the terminal ventricular deflexion has ended, as in Fig. 66, where the cardiac rate is 131 per minute.

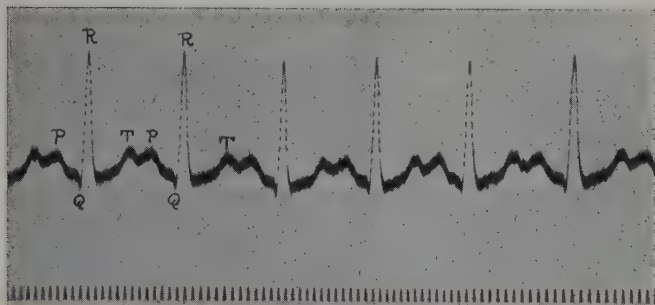


FIG. 66.—SINUS TACHYCARDIA.

The rate of the whole heart is 131 per minute (Derivation III).

The recognition of sinus tachycardia is usually easy. The rate of the pulse is accelerated, though it seldom attains 140, and varies with posture, emotion and exertion. Its rhythm is regular and, unless there is fever or grave disease of the myocardium, the rate waxes during inspiration and wanes during expiration.

SINUS BRADYCARDIA

When the rate of stimulus production in the sinus node is unduly slow, and the whole heart responds to each sinus stimulus, the condition is known as sinus bradycardia. In this condition, which is decidedly less common than sinus tachycardia, the rate is less than 60 per minute; it is usually between 50 and 60,¹ and in exceptional instances may be less than 40. Except in rare cases in which it is purely physiological, bradycardia is a result either of fatigue of the sinus node or of its inhibition by excessive vagal tone (vagotonia). Transient slowing of the whole heart can be produced in many persons by pressing firmly upon the right vagus at the

¹ Willius, F. A., *Arch. Intern. Med.*, 1920, xxvi, 630.

level of the cricoid cartilage. Compression of the left vagus is usually less effective in slowing the heart. Similar effects can be obtained by ocular compression, the finger being pressed upon the right or left eye while the patient looks downward.

An infrequent pulse-rate of 55 to 60 is often observed in hale old people, but is seldom constant in young individuals. In a healthy school-teacher of thirty-one, however, we found a sinus bradycardia with a rate of 54 when standing, and of 50 when sitting ; but a rate so infrequent as this is exceptional. The condition is most often observed during *convalescence* from acute febrile diseases, when the pulse-rate may be notably infrequent, and especially after the crisis of acute lobar pneumonia. We have then observed true sinus bradycardia with rates as low as 46 ; and we have seen the rate as low as 48 after influenza.

In most forms of *poisoning* and of *toxæmia* the pulse-rate is fast rather than slow. This is usually true of tobacco poisoning and of jaundice, but we have observed sinus bradycardia with a rate of 40 in one man, and of only 37 in another, both being cases of tobacco poisoning. In one case of jaundice we found the pulse-rate was only 39, although the sinus rhythm was maintained ; the pulse rose to normal rates when the jaundice passed away. Another man, suffering from diabetes mellitus, had sinus bradycardia with a rate of 43. *Increase of intracranial pressure* is another cause of bradycardia, which may therefore be observed in cases of meningitis and of cerebral hæmorrhage or tumour. In one of our cases of cerebral tumour the pulse-rate was only 39, with a sinus rhythm ; the pulse rose to a normal rate after a decompression operation.

In sinus bradycardia the duration of diastole is unduly prolonged, while that of systole remains unaltered. In *jugular tracings* we find the *a*, *c*, and *v* waves in normal succession, and the *a-c* interval is of normal length. There is therefore no evidence of the infrequent rate being due to defect of conductivity. A fourth wave, *h*, is usually shown clearly (Fig. 67). It is when this wave is obvious that the third heart sound is best heard. Both are produced in the same way, namely by the recoil of the auriculo-ventricular valves consequent upon the inrush of blood into the ventricles.

Electrocardiograms present no abnormality except the undue prolongation of diastole.

The only two conditions likely to be mistaken for sinus bradycardia are auriculo-ventricular heart-block, and *coupled rhythm* with a regularly recurring extrasystole. In the latter the pulse may be infrequent, about 40 per minute, but auscultation reveals a ventricular rate double that of the pulse. The ventricular beats occur in couples, of which only the first causes a pulse wave, while the second is followed by a prolonged pause (*see* Chapter VI, p. 106). In *complete heart-block* the ventricular rate is usually as low as 30 or 36, seldom as high as 40 or 45, per minute; the rate remains practically unchanged by breathing, posture, or exertion, and thus differs from sinus bradycardia. The most satisfactory differ-

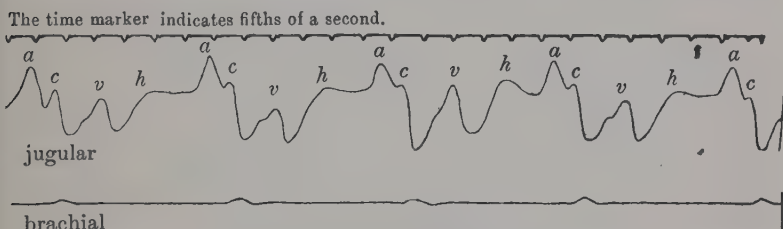


FIG. 67.—SINUS BRADYCARDIA.

The rate of the heart is 51 per minute. From a child, aged 7 years, convalescing from chorea.

entiation of sinus bradycardia from *partial heart-block* and from *nodal bradycardia* is by means of polygraph tracings or electrocardiograms.

SINUS IRREGULARITY

Sinus irregularity is extremely common both in health and in disease, and indicates a varying degree of vagal tone acting upon the sinus node. Careful measurement of normal pulse tracings, even in adults, shows that the regularity of the cardiac action is apparent and not real, although the rhythm appears regular to the finger and even on auscultation. In healthy children and adolescents the arrhythmia is more obvious and seldom escapes notice when the pulse is felt. Children laugh and cry on slight provocation; their temperature rises to inordinate heights from trivial causes;

their breathing when at rest and during sleep is often irregular; and the rhythm and rate of the pulse are also unstable. The most pronounced sinus irregularity is observed in the normal dog's heart.

In sinus irregularity the heart's rate is accelerated during inspiration and retarded during expiration. The maximal irregularity is associated with irregularity of the respiratory phases. The irregularity becomes more obvious during slow deep breathing, is often found to correspond with voluntary irregularity of breathing and to disappear wholly or in part when the individual breathes frequently and regularly, or holds his breath. The association is clearly seen in Fig. 112. At the commencement of the tracing, during quiet breathing, the pulse is notably irregular, long diastoles occurring before each inspiration. When voluntary apnoea is produced the long diastoles are more frequent; but when the respirations become more frequent diastole is shorter and the pulse is much more regular.

Sinus arrhythmia is most obvious in children and in adolescents, and is therefore sometimes known as the "juvenile" form of cardiac irregularity. It may often be observed in adults up to the age of thirty or forty years, but after middle life it becomes less common and less obvious. It is often well seen when the heart is beating slowly after an acute febrile disease, or in neurasthenic or neurotic patients, even though the cardiac rate is notably accelerated.

Graphic records show that the duration of each systole is constant, whereas that of diastole becomes successively shorter or longer according to the phase of respiration. The auriculo-ventricular intervals, *P-R*, or *a-c*, remain constant irrespective of the length of the antecedent diastole. Thus, in Fig. 68, from a healthy lad, the first *P* is obvious; the second begins before the antecedent *T* is completed; the third and fourth coincide with *T*, because of the cardiac acceleration; the fifth and subsequent *P* deflexions, when the cardiac rate lessens, are again clear.

The diastolic pause may be exactly twice the normal length (Fig. 69) (*see* sino-auricular block, p. 140), or may be shorter or longer than this. In exceptional instances the diastolic pauses last for two seconds or more, and the association of

the long pause with the expiratory phase of respiration may be lacking. One of the most striking examples of this nature that we have observed was the occurrence of frequent pauses of 1.7 to 2.0 seconds duration in a man, aged fifty-one, affected with aortic incompetence of rheumatic origin. He had been

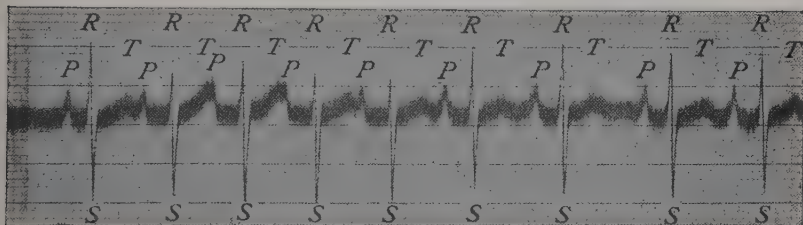


FIG. 68.—SINUS IRREGULARITY. DERIVATION II.
From a healthy individual, aged 20.

taking moderate doses of digitalis for a fortnight, and the sinus irregularity was clearly due to vagal stimulation by that drug. In a few cases we have observed syncopal attacks, due to prolonged diastolic arrest of the whole heart, such as have been graphically described by Laslett,¹ but in these cases there is a sino-auricular block (*see* p. 140).

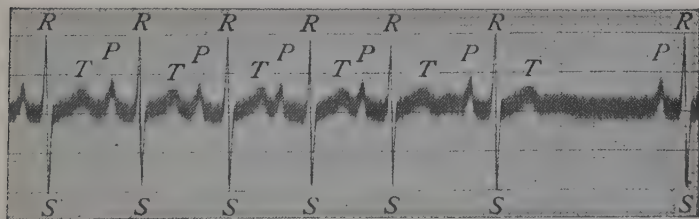


FIG. 69.—SINO-AURICULAR BLOCK. DERIVATION II.

In Cheyne-Stokes, and in cyclic, breathing the pulse-rate often varies during the different respiratory phases (Fig. 193), but the association is not constant, for the pulse-rate during apnœa may be unaltered, or more frequent, or less frequent, than during dyspnœa.

It is important to recognize the nature of sinus irregularity

¹ Laslett, E. E., *Quart. Journ. of Med.*, 1908-9, ii., 347.

because it has no serious significance. During the war many soldiers, in whom the heart was perfectly healthy, were sent to hospital, or invalided out of the army, because they presented a sinus irregularity ; and in civil life we know of no physical sign in relation to the heart which is more often misunderstood and misinterpreted as a sign of disease. Sinus irregularity is seen in health and in disease, but the heart loses this normal irregularity in the most grave forms of cardiac disease involving widespread acute or chronic myocarditis and severe heart failure. Consequently in acute endocarditis, and in rheumatic fever, diphtheria, influenza, and other acute infective diseases, in which myocarditis is liable to supervene, the retention of a sinus irregularity is a favourable sign.

Sinus irregularity calls for no treatment. Digitalis is often administered in the erroneous belief that this irregularity of the pulse is a sign of heart disease. If the drug has any effect in these cases it is mainly that of stimulating the vagus, thereby slowing the heart and rendering the irregularity more obvious. Atropine will render the pulse more regular by paralysing the vagal endings in the heart and thereby increasing its rate, but sinus irregularity is never an indication for the administration of this drug.

Contraction stimuli formed at abnormal sites, namely elsewhere than at the sinus node, are said to be heterotopic or ectopic in origin. They give rise to extrasystoles or to abnormal rhythms, which are discussed in subsequent chapters.

CHAPTER VI

EXCITABILITY

AN extrasystole is a premature and abnormal contraction starting at an abnormal site in the heart. The stimulus for an extrasystole does not arise in the sinus node, but elsewhere, and the resulting contraction is therefore sometimes termed an ectopic beat. The stimulus acts on the heart muscle after its refractory phase has ended, but before the next physiological stimulus from the sinus node has become effective. The auricles alone, or the ventricles alone, may contract prematurely, or both may participate in the contraction. An extrasystole is one of the most frequent forms of cardiac

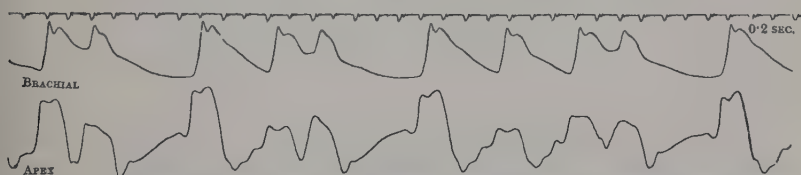


FIG. 70.—VENTRICULAR EXTRASYSTOLES. THE SECOND, FIFTH AND NINTH BEATS ARE VENTRICULAR EXTRASYSTOLES, EACH OF WHICH IS FOLLOWED BY A FULLY COMPENSATORY POST-EXTRASYSTOLIC PAUSE.

irregularity and is readily recognized when feeling the pulse, the regular sequence of beats being interrupted by an “intermission” of the pulse, or by an early beat which is followed by an interval of longer duration than the average.

An extrasystolic pulse wave is always of smaller volume (Fig. 70) and of lower pressure (Fig. 77) than the normal beats. Because of the short diastole preceding an extrasystole, the ventricle is not well filled with blood and its output is therefore lessened; its contractility is not fully restored and the maximal contraction possible is therefore of lessened power; while the aortic blood pressure is still at a higher

level than is reached after an interval of normal length. The prolonged diastole after an extrasystole is termed the post-extrasystolic, or compensatory, pause; and the first beat after that pause is termed the post-extrasystolic beat. On account of the prolonged diastole this beat is, as a rule, a larger, stronger and better sustained wave than those succeeding it.

An extrasystole may start in any portion of the heart. According to their site of origin, we recognize three varieties, namely, ventricular, auricular and nodal extrasystoles. In most cases the extrasystole is a single one, interrupting the sinus rhythm for one beat and recurring from time to time; in rarer instances there is a series of extrasystoles, only the last of the series being followed by a compensatory pause.

Frequency of Extrasystoles.—With the exception of sinus arrhythmia, extrasystoles are the most common of all forms of cardiac irregularity. Hardly a day passes but we observe them, whereas auricular fibrillation is seen once or twice a week, auricular flutter once a month, and heart-block once or twice a year. In a series of 2,425 soldiers admitted to hospital for disorders of the heart, we observed extrasystoles in 6.6 per cent., auricular fibrillation in 0.1 per cent., and heart-block in none.

Etiology.—Experimentally, extrasystoles can be produced in various ways: by direct application of mechanical, thermal, electrical, or chemical stimuli to the heart; by clamping the aorta, the pulmonary artery, or the great veins; by ligation of a branch of the coronary artery; by the injection of digitalin, adrenalin, aconitine, muscarine, and physostigmine; and by stimulation of the sympathetic, of the vagus, or of both together. Extrasystoles can be produced in hearts which are isolated or whose nervous connections are wholly severed; consequently while the cause may be extra-cardiac it may also be in the heart itself. The direct application of stimuli to excitable muscle naturally provokes a contraction; the obstruction of a great artery produces over-distension of the cavities behind the block, and provides a mechanical stimulus; ligation of a coronary branch interferes with the nutrition of the part concerned by reason of the ischæmia

which follows ; and the injection of drugs involves a chemical as well as a mechanical stimulus.

Extrasystoles occur at all ages, though they are least common in childhood, and are comparatively rare in persons below the age of thirty. In 70 per cent. of all cases the patient's age is over thirty-nine. The age and sex incidence in 226 of our cases is shown in the following table.

TABLE SHOWING THE AGE AND SEX INCIDENCE IN 226 CASES WITH EXTRASYSTOLES.

Age	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80 and over.	Total.
Ventricular Extrasystoles :									
Males	3	9	19	26	25	25	5	—	112
Females . . .	1	5	9	18	6	8	4	1	52
Auricular Extrasystoles :									
Males	—	1	1	3	2	6	1	—	14
Females . . .	1	1	1	1	—	2	1	—	7
Nodal Extrasystoles :									
Males	—	3	3	5	4	1	5	—	21
Females . . .	1	—	1	2	2	1	—	—	7
Ventricular and Nodal or Auricular :									
Males	—	—	—	1	1	1	2	—	5
Females . . .	1	2	1	—	1	—	3	—	8
Total	7	21	35	56	41	44	21	1	226

Extrasystoles are observed in persons in all ranks of life, in the poor and in the rich, in those who are brain-workers and in those engaged in manual labour. Any occupation or disease, such as syphilis, which tends to induce *arterial disease* predisposes to extrasystoles. They occur in persons whose arteries are apparently healthy and whose blood pressure is not excessive ; while others with gross arterial disease may never have an extrasystole. Nevertheless there is, as a rule, a close relation between this form of irregularity and arterial degeneration. Unless due to nervous or toxic causes, or to acute myocarditis, extrasystoles are comparatively seldom

observed in persons under forty. After that age the arteries are losing their elasticity and becoming thickened, and extrasystoles are common. In patients over middle age whose blood pressure is rather high, and whose arteries are apparently healthy, extrasystoles which are not due to any obvious cause are often the initial signs of chronic disease of the coronary arteries. In an otherwise perfectly healthy man, whose arteries were soft and whose urine was normal, extrasystoles were first noticed at the age of fifty-one; twelve years later he died of arterio-sclerosis, heart failure and uræmia. Another patient developed extrasystoles at the age of forty-five, and when sixty began to suffer from angina pectoris.

Extrasystoles are most common in patients with *myocardial disease*, the result of chronic disease of the coronary arteries, or of acute rheumatic *endocarditis*, especially of the mitral valve, and they are frequently present in the later stages of acute endocarditis at a time when submiliary nodules are being formed. It seems probable that in many cases some local lesion has produced a local hyper-excitability which induces premature contractions in response to stimuli of minimal degree.

Elevation of the pressure within the heart may evoke extrasystoles; they are often due to this factor, a prior lesion having rendered some part unduly irritable. Extrasystoles are often observed in patients with *cardiac dilatation and failure*. They may have had chronic valvular disease for years, and only develop extrasystoles when they become breathless or dropsical. When these patients improve, and the intracardiac pressure is lowered, the extrasystoles may lessen in frequency or disappear. The pressure within the chambers of the heart is constantly varying with the varying phases of systole and diastole, with the respiratory movements, and with physical exertion; in persons in whom extrasystoles occur spontaneously they can often be produced at will by slight physical exertion or by the cessation of respiration. Mere change of posture may excite or abolish extrasystoles. A woman, aged sixty-five, with thickened arteries, emphysematous lungs and a rough first sound at the cardiac apex, had numerous extrasystoles immediately after she walked into the room, but they disappeared when she lay on a couch.

Another woman, aged fifty-five, who had an ovarian tumour extending up to the level of the umbilicus, chronic phthisis, thick arteries and cardiac hypertrophy, had no extrasystoles when recumbent, but when she sat up in bed, they immediately appeared.

In other cases, however, the increased excitability is certainly of functional origin. Experimentally, extrasystoles can be excited by stimulation of the sympathetic, of the vagus, or of the two combined, and clinical experience teaches that in some persons extrasystoles are certainly of *emotional* origin. Fright, anxiety, fear, joy, and other emotions often cause extrasystoles by stimulation of the sympathetic. In *fever* the cardiac rhythm is unstable and the pulse rate alters notably from causes, for example, emotion or exertion, which would produce little change in a healthy individual. Extrasystoles are not uncommon in febrile diseases, and may be due to increase of excitability by the fever or toxæmia rather than to local lesions within the muscle, though these are often present even in the absence of endocarditis. Extrasystoles, moreover, may occur in persons who are suffering from alcoholic or nicotine *poisoning*, from gastric or intestinal indigestion of varying origin, or from the metabolic disturbances associated with the presence of uric acid or oxalates in excess in the urine; and the cardiac irregularity tends to disappear when the intoxication is remedied. In these diseases the blood pressure is often elevated, and as extrasystoles can be produced by reflex disturbance of the vaso-motor centre the association is extremely suggestive. Extrasystoles are sometimes clearly due to intolerance of tobacco, as in a lawyer aged forty-five in whom a single cigarette invariably induces them.

Synchronous Contraction of auricles and of ventricles may evoke an extrasystole. The synchronous contraction may have occurred because of partial or of complete auriculo-ventricular block. For example, in Fig. 71, the third ventricular beat coincided with the fifth auricular beat; the left ventricle was unable to expel its contained blood through the incompetent mitral valve, and the sudden rise of pressure during the next ventricular diastole evoked a ventricular extrasystole. Similarly an auricular extrasystole may follow, and be the result of, a ventricular extrasystole in which all

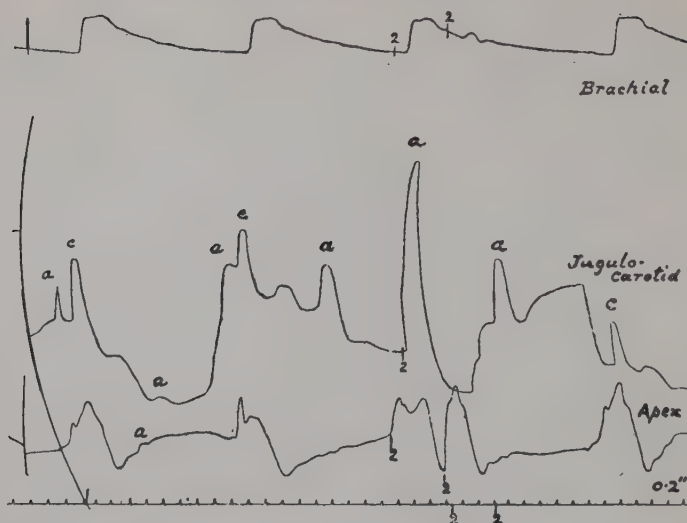


FIG. 71.—COMPLETE AURICULO-VENTRICULAR BLOCK.

The third ventricular contraction coincides with an auricular contraction, and is quickly followed by a premature ventricular beat.

the chambers of the heart contracted synchronously (see Fig. 97).

An undue *prolongation of diastole* may likewise be followed by an extrasystole, as is shown in Fig. 72, obtained from a

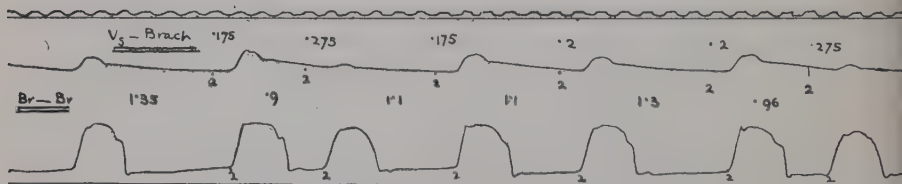


FIG. 72.—AURICULAR FIBRILLATION.

The ventricular rhythm is irregular. The contractions succeeding the longer diastoles are rapidly succeeded by premature beats.

patient whose auricles were in fibrillation, and in Fig. 73, where the prolongation of diastole was readily produced by pressing upon the left vagus nerve.

Symptoms.—An extrasystole does not, as a rule, cause any subjective symptoms. It occurs without the individual

being aware of it. If patients are conscious of extrasystoles they complain of occasional thumping, bumping, flapping, wobbling or palpitation of the heart. Those who are conscious of the post-extrasystolic pause say the heart occasionally stands still or stops ; in a few, the long diastolic pause causes

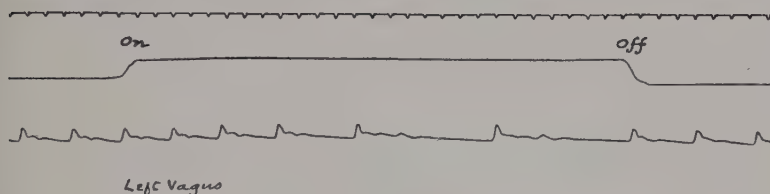


FIG. 73.

The upper curve shows the period of pressure upon the left vagus nerve. Diastole is lengthened by this procedure, and extrasystoles occur in consequence.

momentary giddiness. Others feel the strong post-extrasystolic beat as a throb in the chest, in the episternal notch, in the vessels of the neck, or in the ears. Some patients, and particularly those of a nervous temperament, are acutely conscious of each extrasystole ; it may be referred to as a momentary pain in the chest. In a miner aged forty-two, each extrasystole was attended by twitching of the fingers, but any disturbance of this nature is rare. A short series of extrasystoles may be felt as a "fluttering" sensation within the chest ; a long series may occasion a prolonged attack of palpitation (*see p. 212*).

The Arterial Pulse.—When an extrasystole occurs we notice an "intermission" of the pulse, or a small, weak, premature wave followed by a long pause, which is ended by

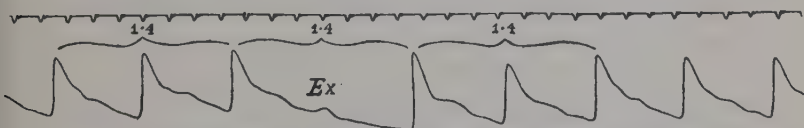


FIG. 74.—AN EXTRASYSTOLE (Ex) FOLLOWED BY A FULLY COMPENSATORY POST-EXTRASYSTOLIC PAUSE.

a strong beat (Fig. 74). If the extrasystole is markedly premature, the corresponding pulse wave may be too weak to be palpable, or the ventricular contraction may be so pre-

mature and feeble that it fails to open the aortic valve (Fig. 75). But in these circumstances the long diastole is very noticeable. If we are counting the pulse beats, and the first one after the intermission occurs precisely at the moment when it is due, the post-extrasystolic pause is fully compensatory,

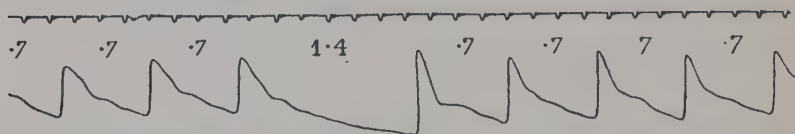


FIG. 75.—THE EXTRASYSTOLE DOES NOT CAUSE A PULSE WAVE, BUT THE POST-EXTRASYSTOLIC PAUSE IS FULLY COMPENSATORY.

and the extrasystole started in the ventricle. But if the pause is not fully compensatory and the post-extrasystolic beat occurs before it is due, the extrasystole is almost certainly auricular or nodal in origin. This easy method of differentiating ventricular from supraventricular (auricular and nodal) extrasystoles is fairly reliable.

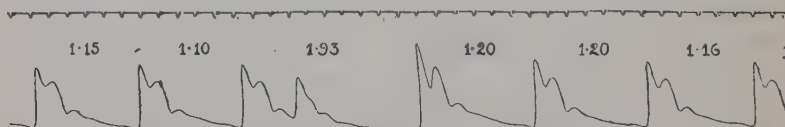


FIG. 76.—THE FOURTH BEAT IS AN EXTRASYSTOLE WHICH IS FOLLOWED BY A PAUSE THAT IS NOT FULLY COMPENSATORY.

If extrasystoles occur while the blood pressure is being determined by the auscultatory method, they are easily recognized when the pressure within the armlet is a little below the systolic pressure. The contrast which is then presented between the loud arterial sound accompanying each normal beat and the intermission, or faintness, of the arterial sound accompanying an extrasystole is very distinct. For example, if the systolic pressure of each normal beat be 130 mm. Hg., and the pressure within the sphygmomanometer be set at 120, an extrasystole which does not raise the blood pressure above 100 mm. Hg. will not cause an audible arterial sound.

By palpating the apex impulse, or preferably by auscultating the heart, while we are feeling the radial pulse, the extrasystole becomes more obvious. It causes either two sounds,

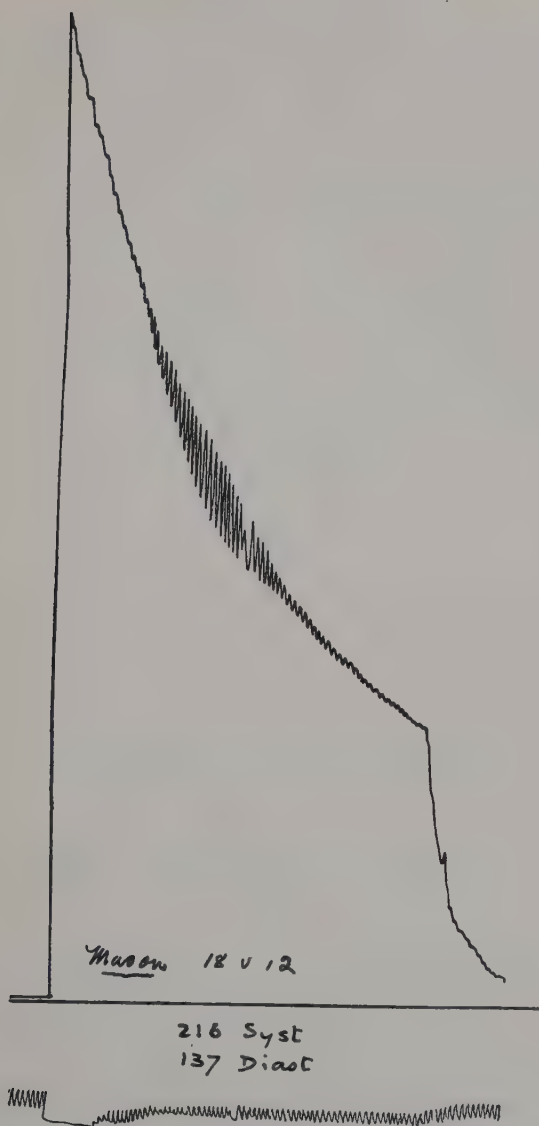


FIG. 77.—SPHYGMOMANOMETRIC TRACING, SHOWING THE LOWER PRESSURE OF THE EXTRASYSTOLIC BEAT.

or one, according as the semilunar valves are opened, or not. In the latter case there is no extrasystolic pulse wave, but merely an intermission (Fig. 75).

If we watch the jugular pulsation at the root of the neck while feeling the radial pulse and auscultating the heart, we often see an unusually large wave in the jugular vein each time there is an extrasystole (Fig. 78). The reason for this large wave is that during the extrasystole the auricles contract synchronously with the ventricles and cannot empty their contained blood into the latter because the auriculo-ventricular orifices are closed; consequently there is either a wave of increased pressure in, or an actual regurgitation of blood into the great veins. This large jugular wave is seen in nodal extrasystoles, both auricles and ventricles contracting prematurely and synchronously; and in ventricular extrasystoles when the premature contraction of the ventricles

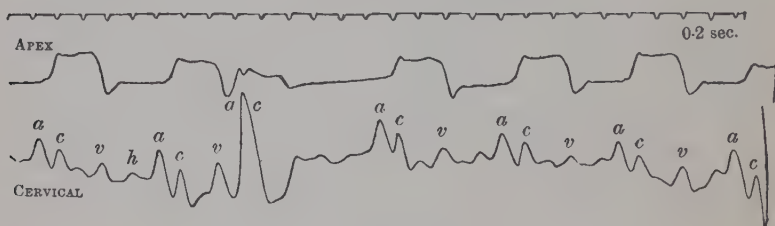


FIG. 78.

The third beat is a nodal extrasystole, in which the auricles and ventricles contract prematurely and synchronously. From a man, aged forty-two, who had acquired syphilis fifteen years previously.

coincides with a rhythmic auricular beat. It is not seen in auricular extrasystoles, because the auricular contraction precedes the ventricular.

By simultaneous palpation of the radial pulse, auscultation of the heart, and inspection of the jugular veins, we can differentiate, with a fair degree of accuracy, between auricular, nodal, and ventricular extrasystoles. If the post-extrasystolic pause is fully compensatory the extrasystole is almost certainly ventricular. If that pause is not fully compensatory the extrasystole is probably supraventricular; it is nodal if there is an unusually large jugular impulse during the extrasystole; if the wave is not large the extrasystole is auricular.

Extrasystoles usually recur at varying intervals. In some patients they are infrequent, the sinus rhythm being unbroken for long periods varying from minutes to days. Other patients

present frequent extrasystoles; several may occur in the course of a minute, or their number may be so great and their



FIG. 79.—VENTRICULAR EXTRASYSTOLES (ES) RECURRING REGULARLY AFTER EACH NORMAL BEAT.

type so varied that the pulse resembles that of auricular fibrillation. An extrasystole may recur regularly after each



FIG. 80.—A VENTRICULAR EXTRASYSTOLE (ES) AFTER EVERY SECOND NORMAL BEAT.

The post-extrasystolic pause is fully compensatory.

physiological beat (Fig. 79) constituting a bigeminal pulse or a coupled rhythm (*see* p. 106), after every second beat



FIG. 81.—A VENTRICULAR EXTRASYSTOLE (ES) AFTER EVERY THIRD NORMAL BEAT.

The post-extrasystolic pause is fully compensatory.

(trigeminal pulse, Fig. 80), or after every third, fourth or fifth beat (Fig. 81, 82). Further information regarding the

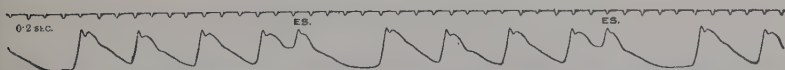


FIG. 82.—A VENTRICULAR EXTRASYSTOLE (ES) AFTER EVERY FOURTH NORMAL BEAT.

The post-extrasystolic pause is fully compensatory.

cardiac mechanism during an extrasystole can be obtained by tracings or by electrocardiograms.

VENTRICULAR EXTRASYSTOLES

About 70 per cent. of all extrasystoles start in the ventricles. The ventricular contraction usually begins before, but may

begin immediately after, a rhythmic auricular contraction, and the two usually coincide. Ventricular extrasystoles seldom interfere with the rhythm of the auricles or disturb the rhythmic production of stimuli in the sinus node. The post-extrasystolic pause is therefore fully compensatory, for

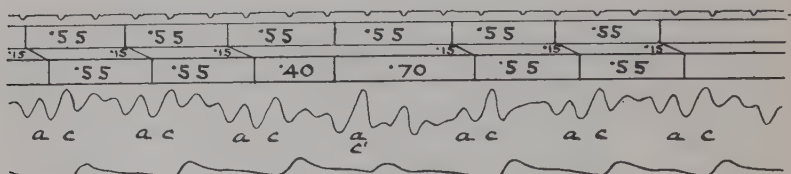


FIG. 83.

The fourth beat in the lower (arterial) curve is a ventricular extrasystole which is followed by a fully compensatory pause. The cervical curve shows the auricular waves, *a*, appearing rhythmically. In the upper part of the figure, below the time record (0.2 second), is a diagram showing the rhythm of the auricles, the *a-c* interval (0.15 second) and the ventricular rhythm.

it is of such a length that, taken in conjunction with the pulse period preceding the extrasystole, it measures exactly double the normal pulse period; and the post-extrasystolic contraction of the ventricles is delayed until the next sinus stimulus, whose rhythm is undisturbed, reaches the ventricles (Fig. 83).

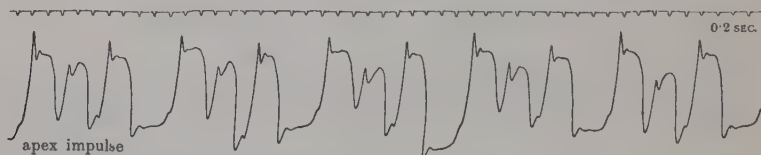


FIG. 84.

The second, fifth, eighth, eleventh and fourteenth beats are interpolated extrasystoles. The beat following each extrasystole occurs precisely when it is due.

In exceptional cases a ventricular extrasystole is "interpolated," occurring so early in diastole that the succeeding sinus stimulus evokes an auricular and a ventricular response¹ (Fig. 84). Interpolated extrasystoles are rarely seen except in cases of auriculo-ventricular block, or when the heart's

¹ Myers, M. M., and White, P. D., *Arch. Intern. Med.*, 1921, xxvii., 503.

rate is for other reasons below sixty per minute. In other exceptional instances, the post-extrasystolic pause is not fully compensatory because an auricular extrasystole succeeds

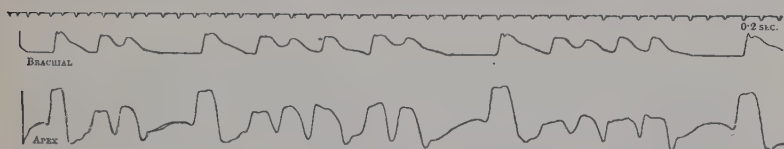


FIG. 85.

The first, second, fourth, fifth, tenth and fifteenth beats are of sinus origin ; the other beats are extrasystoles, single or in series.

the ventricular one and disturbs the sinus rhythm, as in Fig. 97. The reason for the ventricular extrasystole being followed by an auricular one is that the contraction of the auricle and



FIG. 86.—EXTRASYSTOLES (ES) OCCURRING SINGLY, IN PAIRS, AND IN SERIES OF THREE.

From a man aged thirty-four who had suffered from rheumatic fever four years previously, and who presented the signs of mitral incompetence.

of the ventricle having coincided the former is not completely emptied at the end of systole, and being rapidly distended during diastole by the blood flowing quickly into it from the

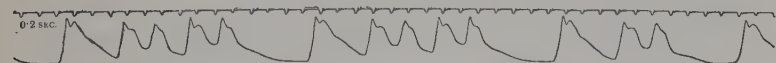


FIG. 87.—MULTIPLE EXTRASYSTOLES.

over-distended venæ cavæ, the auricle is induced to contract prematurely by the sudden increase of pressure within its

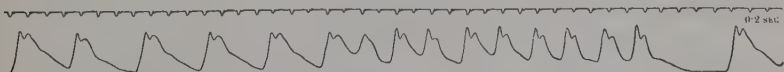


FIG. 88.—After six normal beats there is a series of extrasystoles terminated by a post-extrasystolic pause which is followed at the end of the tracing by a normal beat.

cavity. The auricular extrasystole is therefore a direct result of the preceding ventricular extrasystole.

In other instances, extrasystoles, all of identical form, follow

one another in rapid succession, and only the last of the series is followed by a post-extrasystolic pause. The number of extrasystoles in the series is usually only two or three; rarely there is a paroxysm of extrasystoles, at a rate of from 150 to

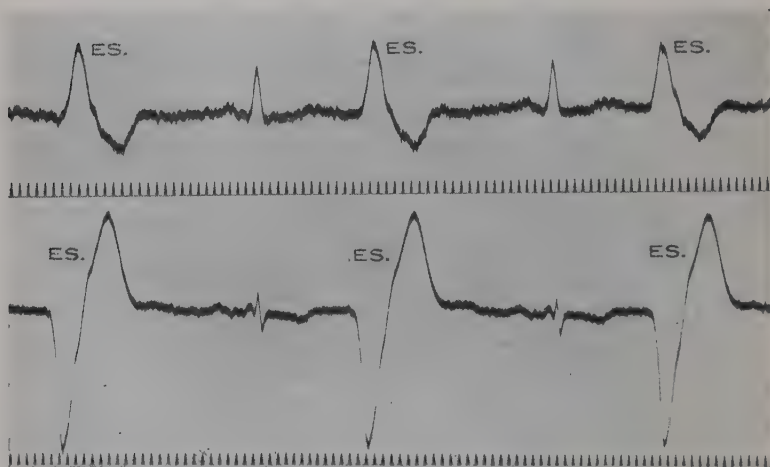


FIG. 89.—LEFT APICAL EXTRASYSTOLES (ES) RECORDED BY DERIVATION *I* IN THE UPPER ELECTROCARDIOGRAM, AND BY DERIVATION *III* IN THE LOWER.

260 per minute, lasting for a few minutes, causing palpitation, and ending in a post-paroxysmal pause before the sinus rhythm is regained (Fig. 88).

In an electrocardiogram the complex of a ventricular extra-

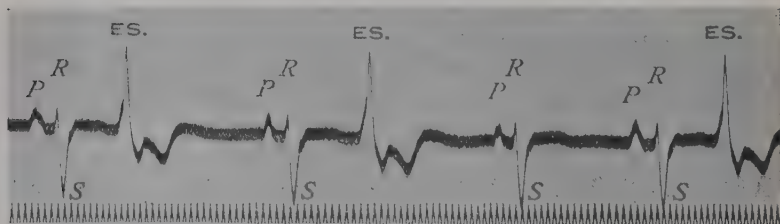


FIG. 90.—VENTRICULAR EXTRASYSTOLES (ES). DERIVATION *II*.

systole is wholly atypical as contrasted with the normal, for the stimulus originates at an abnormal site, probably in the arborization of the auriculo-ventricular bundle, is transmitted

in an abnormal manner through the ventricles, and causes the various parts of their musculature to pass into activity in abnormal sequence. An extrasystole is thus not merely a premature, but a wholly abnormal, contraction. The char-

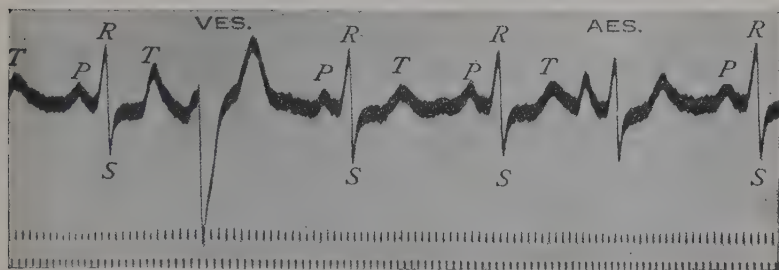


FIG. 91.—SHOWING A VENTRICULAR (VES) AND AN AURICULAR (AES) EXTRASYSTOLE. FROM A MAN AGED FORTY-NINE, SUFFERING FROM SYPHILITIC AORTITIS. DERIVATION II.

acteristic ventricular complex is diphasic and of larger amplitude than that of a physiological beat (Figs. 89, 91); in the latter respect it is in striking contrast to the feeble pulse wave transmitted into the arteries (Fig. 164). The duration of the ventricular complex is on an average 0.373 second, this

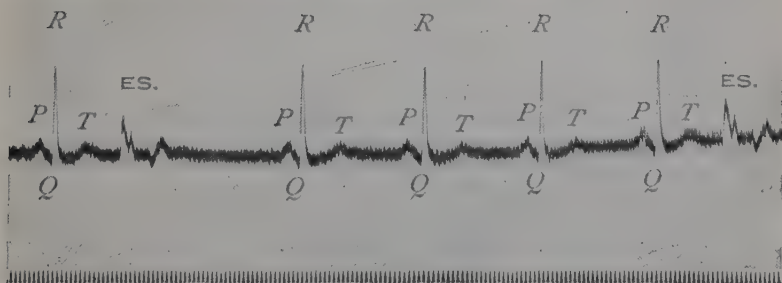


FIG. 92.—VENTRICULAR EXTRASYSTOLES (ES), EACH WITH AN ABERRANT COMPLEX. DERIVATION III.

being an increase of 0.05 second over the average normal ventricular complex.¹

Two varieties of ventricular extrasystole are recognized: (1) Left apical extrasystoles in which, as recorded by

¹ Cowan, J., and Ritchie, W. T., *Lancet*, 1920, ii., 743.

Derivation *I* (right and left hands), the initial deflexion is first upward and then downward (Fig. 89), and (2) Right basal extrasystoles in which, by the same derivation, the

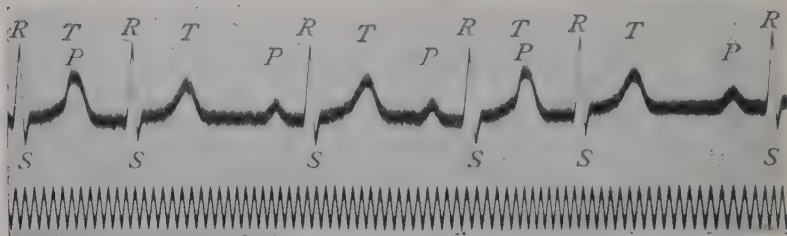


FIG. 93.

The first, third, fourth and last beat are of sinus origin. The second and fifth beats are auricular extrasystoles, the deflexion *P* coinciding with the deflexion *T* of the preceding beat, and the *P-R* interval being prolonged. From a man aged twenty-one.

deflexion is first downward and then upward. When recorded by Derivation *III* (left hand and left foot) the form of the complex is reversed. Consequently a left apical extrasystole

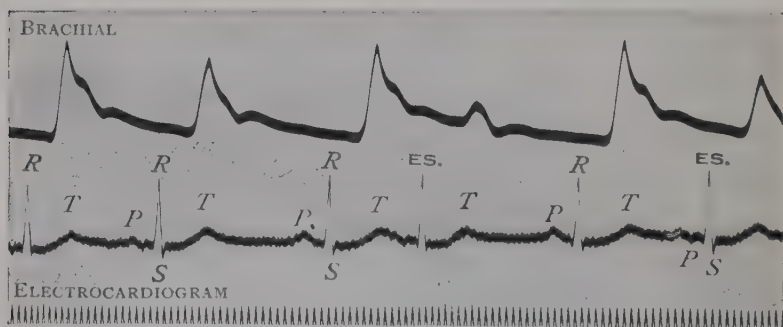


FIG. 94.

The second and sixth beats are supra-ventricular extrasystoles with abnormal deflexion, *P*. The fourth beat (ES) is also a supra-ventricular extrasystole, but the deflexion *P* is not seen. By Derivation *II* from a man aged seventy-two, suffering from dyspnoea and dropsy.

by Derivation *III* yields a complex like that of a right basal extrasystole by Derivation *I*, and the latter by Derivation *III* presents the same form as does the first variety by Derivation *I*.

In our experience it is not possible accurately to determine, solely from the clinical features of the case, whether the extrasystoles are left apical or right basal. A man aged forty-eight, suffering from chronic bronchitis, chronic nephritis and dropsy,

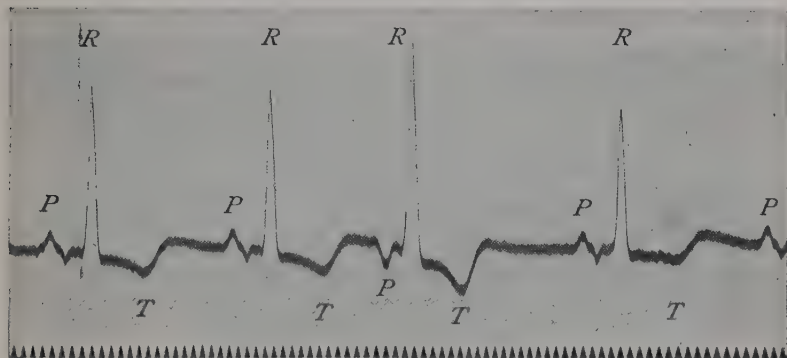


FIG. 95.—NODAL EXTRASYSTOLES.

The third beat is a nodal extrasystole with inverted *P* deflexion. Derivation *III*.

had left apical extrasystoles; while in another man, aged fifty, and with similar symptoms, they were right basal. A man aged seventy-three with arteries like pipe stems had

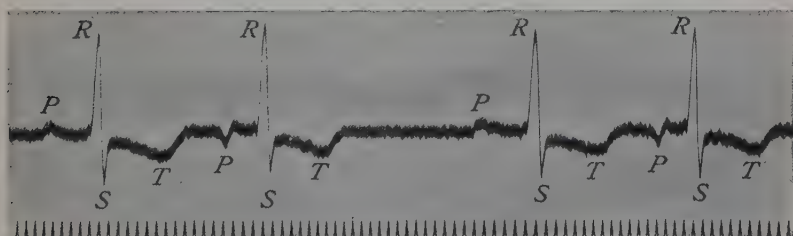


FIG. 96.—COUPLED RHYTHM.

The first and third beats are of sinus origin, but the *P-R* interval is prolonged (0.17 second). The second and fourth beats are nodal extrasystoles with inverted *P*. Derivation *II*, from a man aged sixty-nine.

left apical extrasystoles, whereas they were right basal in another, aged sixty-nine, whose arteries were also calcareous. In association with the various valvular lesions, aortic and mitral, the ventricular extrasystoles may be of either variety.

About 13 per cent. of patients presenting ventricular extrasystoles have both varieties.

AURICULAR EXTRASYSTOLES

The premature contraction begins in the auricle and this is followed by a premature ventricular contraction (Fig. 93). The contraction probably starts in the vicinity of the coronary sinus, or at some other site in the auricular septum or walls.

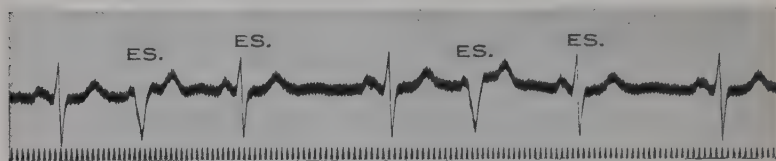


FIG. 97.

After each normal beat there are two extrasystoles (ES), the first being ventricular, the second being supraventricular in origin. Derivation II, woman aged thirty-five suffering from mitral stenosis and incompetence, chronic pericarditis and chronic nephritis.

In electrocardiograms the auricular deflexion, *P*, may be upward or inverted; the more it differs from the normal the further removed from the sinus node is the initial site of contraction. The stimulus is transmitted to the ventricles along the normal path, namely the auriculo-ventricular bundle, and the premature ventricular contraction therefore yields a normal complex. In exceptional instances the ventricular

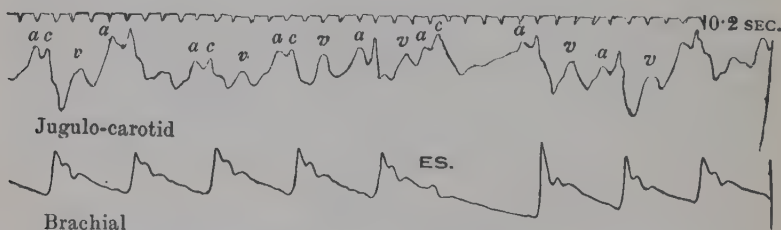


FIG. 98.—POLYGRAPH TRACINGS SHOWING AN AURICULAR EXTRASYSTOLE (ES).

complex differs somewhat from the normal, and is sometimes termed an aberrant complex, to distinguish it from the more atypical complex of a ventricular extrasystole.

An auricular extrasystole almost invariably disturbs the

sinus rhythm, the stimulus material in the sinus node being "exploded" prematurely, and the post-extrasystolic pause is therefore not fully compensatory. Any exception to this rule is unusual. As the diastole preceding the extrasystole is short, conductivity has not been fully restored, and the interval between the auricular and ventricular contraction is prolonged. The presphygmic interval and the transmission time are also necessarily increased, consequently the *a-c* interval exceeds the normal 0.15 to 0.20 second (Fig. 98).

NODAL (AURICULO-VENTRICULAR) EXTRASYSTOLES

In these there is a premature and synchronous contraction of the auricles and of the ventricles. The stimulus probably arises in the auriculo-ventricular node, passing backward to the auricles and onward to the ventricles. The ventricular complex is normal. If the auricles begin to contract first, their deflexion is clearly atypical and usually inverted, and the auriculo-ventricular interval is decidedly shorter than the normal 0.14 second (Figs. 95, 96). If the ventricles begin to contract before the auricles, the auricular deflexion is masked and distorted by the initial ventricular deflexions (*see* Fig. 167, Chapter XII).

In jugular tracings the auricular and carotid waves are both premature, the former preceding or succeeding the latter, and they are either separated by an extremely short interval, or mingled to form one wave (Figs. 78, 99), which is larger than usual because the auricular contraction is occurring at a time when the auriculo-ventricular valves are shut, as described on page 96.

Nodal extrasystoles are somewhat more frequently observed than auricular (*see* Table on p. 89), and both are more frequent in men than in women. The duration of ventricular systole in nodal extrasystoles is on an average 0.009 second shorter than, whereas that of auricular extrasystoles is equal to that of the normal beats.

As a general rule, the type of extrasystole in any individual, whether it be ventricular, nodal, or auricular, remains constant. Only 12 per cent. of our cases have presented auricular or nodal as well as ventricular extrasystoles; all these patients

had organic disease of the heart, and the majority are known to have died within a few months after coming under observation.

In exceptional cases the heart's rate is retarded, in others it is accelerated for a few beats either before or after an extrasystole. These alterations are probably an expression of change in vagal tone. The post-extrasystolic pause is some-

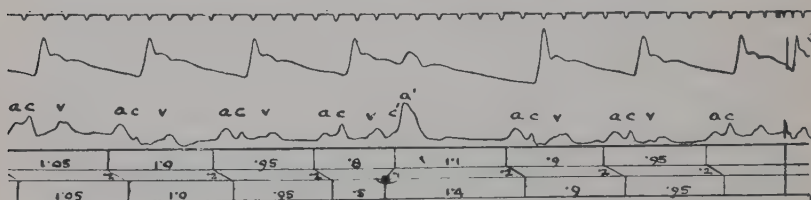


FIG. 99.—NODAL EXTRASYSTOLE, ARTERIAL AND CERVICAL CURVES.

The fifth pulse wave is a nodal extrasystole in which the ventricular contraction precedes the auricular contraction.

times more than fully compensatory, this being due to an inhibitory effect of the extrasystole on the heart. Alternation of the pulse if present always becomes more obvious after an extrasystole, or alternation may be observed only in the first few beats after an extrasystole and gradually passes away. This feature should always be looked for carefully, because it indicates a grave prognosis (*see* p. 150).

COUPLED RHYTHMS

A ventricular extrasystole recurring regularly after each physiological beat represents the most common form of coupled rhythm, or bigeminal pulse (Figs. 79, 100, 101). In a coupled rhythm the contractions occur in pairs, the interval between the two beats of each pair being shorter than that which separates the second beat from the first of the succeeding pair. In exceptional cases the coupling of the cardiac beats is the expression of a sinus irregularity, in which all the chambers of the heart participate. This form of coupled rhythm has no pathological significance. For example, we observed this rhythm ten years ago in a medical student who has since enjoyed excellent health and who was on active service for several years during the war. Another infrequent form of

coupled rhythm is that resulting from auriculo-ventricular block, when every third sinus stimulus fails to reach the ventricles (*see* p. 118).

In all other forms of coupled rhythm, however, the second beat of each couple is an extrasystole. Henschen¹ pointed out that coupled rhythm occurs most frequently in mitral disease with a feeble heart and a pulse rate which has been rendered infrequent by full doses of digitalis. The theory which he advanced to explain the disorder of rhythm is applicable to cases in which the second beat of each couple originates in the auricles. Investigating the problem in 1910,² we found that extrasystolic coupled rhythm is almost invariably associated with evidence of cardiac weakness, and very often with mitral incompetence. Cases in which coupled rhythm is long maintained are usually suffering from chronic heart failure; but in others presenting a coupled rhythm for shorter periods the degree of cardiac failure may be equally severe. The ventricular rate is usually of moderate frequency.

The extrasystole forming the second beat of each couple occurs at a constant, and brief, interval after the first beat, and is usually but not invariably a ventricular extrasystole (Figs. 100, 101). It may send a pulse wave into the arteries, or may fail to open the aortic valve. In most cases the auricles are in fibrillation and the length of the diastolic period after the extrasystoles is wholly inconstant (*see* p. 191, and Fig. 164).

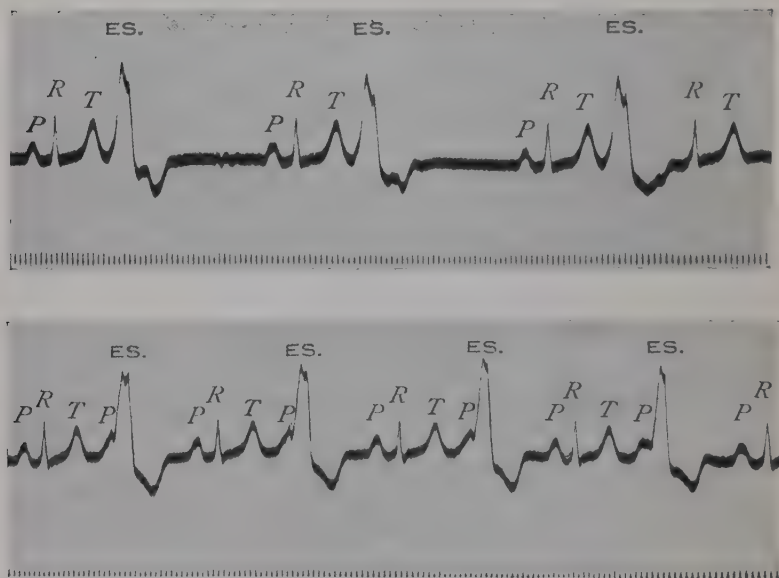
In other cases, however, the rhythm of the heart is dominated by the sinus node, and the length of diastole after each regularly recurring extrasystole is constant. In this form of coupled rhythm we assume that there is an irritable site in the ventricle and that the mitral valve is incompetent. If on any one occasion, from lengthening of diastole or synchronous contraction of auricle and ventricle, the latter is imperfectly emptied at the end of systole, the diastolic influx from the auricle will rapidly produce so great a degree of distension that the irritable area in the ventricular wall initiates a premature contraction. The prematurity of the contraction insures complete emptying of the ventricle, for

¹ Henschen, *Mitteil. a. d. med. Klinik zu Upsala*, 1898, i.

² Cowan, J., and Ritchie, W. T., *Quart. Journ. of Med.*, 1910-11, iv., 55.

the auricle is as yet relatively empty, and the systolic reflux is, in consequence, full ; and no over-distension of the ventricle will occur before the succeeding sinus stimulus arrives. The lengthened diastole which thus ensues again entails over-distension of the ventricle, despite its contractility having improved during the longer period of rest, and the chamber is again imperfectly emptied ; and the process may thus be continued indefinitely.

It is comparatively rare for the second beat of each couple



FIGS. 100, 101.—COUPLED RHYTHM DUE TO VENTRICULAR EXTRASYSTOLES (ES) RECURRING AFTER EACH NORMAL BEAT. DERIVATION II.

to be a regularly recurring auricular or nodal extrasystole (Figs. 96, 102), but this may be observed in cases of mitral incompetence having an irritable focus in the auricular wall. If the auricle becomes over-distended by prolongation of diastole or synchronous contraction of ventricle and auricle, the latter will be unable to empty itself completely. With the succeeding ventricular systole it will rapidly be over-distended by the reflux of blood from the left ventricle, and the excessive intra-auricular pressure will excite a premature

contraction in auricle, followed in due time by a ventricular systole. As this ventricular contraction is premature, the ventricle is incompletely filled, and the regurgitant blood is small in amount, and insufficient to distend the auricle to such a degree as to excite a new contraction, which will only occur when the next sinus stimulus reaches it. The long diastole, however, again entails over-distension, and the coupled rhythm will continue indefinitely until from some external influence (respiration, exertion, etc.) the venous inflow to the auricle is lessened, and with an auricular contraction of sinus origin no over-distension of its cavity coincides.

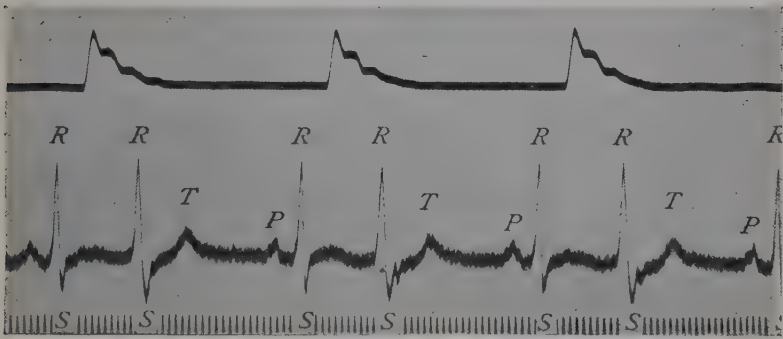


FIG. 102.—SIMULTANEOUS CURVE OF BRACHIAL PULSE WAVE AND ELECTROCARDIOGRAM BY DERIVATION II, SHOWING COUPLED RHYTHM.

The second beat of each pair is a supraventricular extrasystole which causes no arterial pulse wave.

In all these forms of extrasystolic coupled rhythm the two beats of each couple originate at different sites. There are, however, coupled rhythms, those which Wenckebach¹ called "true bigeminy," in which both beats are essentially identical, the stimulus for each originating at the same site. Both beats of each couple may originate in the sinus node, or both may begin in the auriculo-ventricular node while the activity of the sinus node is in abeyance (see Chapter XII).

Extrasystolic coupled rhythm is, as already mentioned, a sign of cardiac weakness. If digitalis or strophanthus are

¹ Wenckebach, K. F., *Die Arrhythmie als Ausdruck bestimmter Funktionsstörungen des Herzens*, Leipzig, 1903, 160.

being administered, a continuous coupled rhythm is an indication to cease or lessen the dose.

Diagnosis.—The recognition of an extrasystole is seldom attended with difficulty, and requires no instrumental aids. *Sinus irregularity* is excluded by noting that the long pause does not coincide with expiration. In *alternation* (Fig. 126) the pulse waves are alternately larger and smaller, but they are either absolutely rhythmic or the smaller wave is delayed, whereas the small wave of an extrasystole is premature. An extrasystolic intermission of the pulse may be mistaken for a failure of ventricular contraction owing to *heart-block*, but on auscultating the heart we hear two sounds, or one, at the moment when the pulse intermits, whereas during the occasional failure of ventricular contraction resulting from heart-block no cardiac sounds are heard. *Auricular fibrillation* is not simulated unless there are very frequent extrasystoles, but even in such cases it will be found, on watching the pulse for a minute or two, that it is absolutely rhythmic from time to time, and this definitely excludes fibrillation.

The differentiation of the varieties of extrasystole from one another is based on the duration of the post-extrasystolic pause, on the character and time relation of the waves in the jugular veins, and on the electrocardiographic curves as described in the foregoing pages.

Prognosis.—The clinical significance of extrasystoles varies in different patients. In the absence of all signs of cardiac insufficiency the prognosis is good, especially if the extrasystoles are clearly due to emotional or toxic causes. A young woman, without any rheumatic history or any symptoms or signs of organic disease of the heart, complained of palpitation due to ventricular extrasystoles as a direct result of the immoderate indulgence in tea and tobacco. Five years later her heart was perfectly normal in every respect. A pensioner who was under our care had many ventricular extrasystoles. His arteries were moderately thick and his Wassermann reaction was strongly positive, but his extrasystoles nearly all disappeared after an encysted empyema was incised and drained. A man aged forty-one, who had

previously been healthy, became confined to bed on account of rheumatic teno-synovitis. This was speedily relieved by salicylates. Two days later occasional ventricular extrasystoles, of which he was unaware, and a transient soft tricuspid systolic murmur were noted. The extrasystoles were never observed subsequently, and ten years later he still enjoys full health and vigour. Another patient, aged forty-eight, with healthy heart and arteries, who was suffering from dyspnœa, cough, hæmoptysis and other symptoms of syphilis of the lung, is now strong and well, eight years after having repeatedly presented ventricular extrasystoles. A man, aged twenty-seven, had an intermittent pulse on November 4, 1882. Next day the cardiac action was regular. Forty years later he was in good health and again, at the age of sixty-eight, proposed to take a fresh life assurance policy.

In young persons without organic disease of the heart the prognosis is good. Of fifty-nine soldiers presenting extrasystoles, thirty-two became fit for full duty on active service within two or three months after their admission to hospital. In old people ventricular extrasystoles are usually of little significance. In a hale old gentleman of seventy-five, they were the only abnormality and he continued to lead an active life, farming his estate, until his death from cancer of the iliac colon five years later. In organic valvular disease ventricular extrasystoles may add nothing to the gravity of the prognosis. A lady aged forty-four with aortic incompetence of rheumatic origin was still actively engaged in office work, though she had been subject to extrasystoles for seven years.

In patients over forty years of age, extrasystoles not due to obvious nervous or toxic causes should be regarded as the earliest indications of sclerosis of the coronary arteries, which will slowly progress, though it may be some years before any further signs become evident.

If extrasystoles are followed by alternation of the pulse, the heart is failing and the prognosis is grave; these patients seldom survive for more than one or at most two years. In acute endocarditis, extrasystoles may be regarded as probable evidence of myocarditis, and are thus of serious import.

As contrasted with ventricular extrasystoles, those of

auricular and nodal origin have, as a rule, a more serious significance, because they are seldom observed except in cases of myocardial or valvular disease who have some limitation of the cardiac reserve power. For instance, a man aged forty-three, the active head of a big business and occupied in his spare time with philanthropic work, had mitral stenosis and incompetence for many years. After an attack of influenza the heart began to fail, and then for the first time auricular extrasystoles developed. Five years later the auricles were fibrillating. Of thirty-two cases with auricular or nodal extrasystoles, seven developed auricular fibrillation, and ten died within a period of two years.

Patients who present both ventricular and supraventricular extrasystoles almost invariably have well-marked heart failure, and do not survive for many months.

Treatment.—The treatment is that of the causal condition. In cases of myocardial or valvular disease with heart failure, treatment is not influenced by the occurrence of extrasystoles. If the extrasystoles point to early arterio-sclerosis, treatment should be directed to retarding its progress. In cases of toxic or reflex origin every effort should be made to discover the source of irritation. Extrasystoles of emotional origin seldom call for treatment, but in some instances the administration of ammonium, or calcium, bromide is indicated.

CHAPTER VII

CONDUCTIVITY

EACH of the stimuli arising rhythmically in the sinus node of a normal heart is transmitted to the auricles and causes them to contract simultaneously. Reaching the auriculo-ventricular node, each stimulus is transmitted thence through the bundle, its branches, and their terminal arborizations, to the ventricles, which contract simultaneously on receipt of the stimulus. The transmission of stimuli through the heart may, however, be defective either from organic or from functional causes. The conducting paths may be the seat of disease, or their capacity to transmit stimuli may be lessened by extrinsic poisons, by toxins, or by vagus stimulation. The defect may cause merely a delay of transmission; it may cause an occasional interruption; or it may amount to a complete block. The defect may lie in any part of the conducting path—in the structures which form the functional bond between the sinus node and the auricles, or between the sinus node and the auriculo-ventricular node, in the latter node, in the bundle, in its branches, or in their arborizations. As defective conductivity is most easily recognized in the auriculo-ventricular bundle, we shall consider this first, and thereafter discuss defects of conductivity in other parts of the heart.

AURICULO-VENTRICULAR CONDUCTIVITY

Defective conduction of stimuli to the ventricles is generally due to gross lesions in the auriculo-ventricular bundle. The earliest experiments, those recorded by Stannius in 1852, showed that after placing a ligature around the auriculo-ventricular junction of the frog's heart the rhythm of the sinus and auricular contractions continued unchanged, whereas

the ventricle, after an initial pause, contracted with a slower and independent rhythm. In Gaskell's experiments (1882) a clamp was applied to the auriculo-ventricular ring in the hearts of the tortoise and frog. When the clamp was gradually tightened, the interval between auricular and ventricular contractions gradually lengthened. With variations in the degree of compression the ventricle could be made to respond to every second, third or fourth auricular contraction, or to remain quiescent for a time ; it then recommenced beating, but independently of the auricles and with a slower rhythm of its own.

In the mammalian heart, Kent, His, Humblet, Hering, Erlanger¹ and many others have demonstrated conclusively that stimuli are conducted to the ventricles by means of the bundle. If this be severed the ventricles, after a pause, beat with a slow, independent, "idio-ventricular" rhythm. If the bundle be compressed gradually by clamping, the interval between auricular and ventricular contractions is first lengthened, then an occasional stimulus to the ventricles is blocked and these chambers miss a beat. Further compression blocks the transmission of every sixth, fifth, fourth, or third stimulus. If the compression of the bundle be increased still further, the ratio of auricular to ventricular contraction changes progressively from 2 : 1, 3 : 1, 4 : 1, etc., until the block becomes complete. The auricles then continue beating rhythmically, whereas the ventricles stand still, and this causing cerebral anæmia, an epileptiform seizure may ensue which ends when the ventricles commence to beat with their slow, independent rhythm. The effects of clamping the bundle are essentially similar to the conditions observed clinically when the bundle is being compressed or severed by disease.

Numerous cases are on record where some degree of auriculo-ventricular block was present during life, and *post-mortem* the bundle was affected by various lesions interfering in greater or less degree with its functional activity. The defect may, however, be functional and exist without gross disease of the bundle. Auriculo-ventricular block may, for example, be produced experimentally by the administration

¹ Erlanger, J., *Journ. of Exp. Med.*, 1906, viii., 8.

of digitalis, adrenalin, or other toxic substance, by vagus stimulation, by asphyxia, and as an anaphylactic reaction.¹ Clinically, a transient block may be induced by compression of the vagus in the neck, or by ocular compression. Defective conductivity can be intensified by stimulating the vagus, for example by compressing that nerve in the neck, or by medicinal doses of digitalis as was first demonstrated graphically by Mackenzie;² whereas the defect can be lessened or abolished for a short time by means of atropine given in doses sufficient to abolish all vagal influence. In complete auriculo-ventricular block, however, the ventricles are beyond the control of the vagus and of the sympathetic; their rate and force of contraction are not influenced by digitalis, atropine or adrenalin.

Three grades of defective auriculo-ventricular conductivity are recognized: (1) Depression of conductivity, (2) Partial block, and (3) Complete block.

(1) Depression of Conductivity

Each stimulus is transmitted to the ventricles, but more slowly than normal. The ventricles continue beating at the same rate as the auricles, but the interval between the auricular and the ventricular contractions is increased beyond the normal 0.14 second. This defect cannot be recognized except by means of tracings or electrocardiograms. In the latter the lengthened auriculo-ventricular interval is revealed by a *P-R* interval exceeding 0.14 or 0.15 second (Figs. 103, 104).

In jugular tracings the lengthened auriculo-ventricular interval is represented by an *a-c* interval measuring more than 0.2 second. In the normal individual this interval measures 0.15 to 0.20 second, being slightly shorter when the pulse rate is frequent. During the *a-c* interval five events occur: (1) The contraction of the auricles; (2) the passage of the stimulus to the ventricles; (3) the latent period before the ventricles contract; (4) the presphygmie

¹ Auer, J., and Robinson, G. Canby, *Journ. of Exp. Med.*, 1913, xviii., 450.

² Mackenzie, J., *Brit. Med. Journ.*, 1905, i., 587.

interval; (5) the transmission of the aortic wave to the carotid artery. The auricular contraction begins 0.04 second before the appearance of the *a* wave in the jugular tracing (see Fig. 62), whereas the average period between the commencement of ventricular systole and the appearance of the carotid wave is 0.066 second. The pre-sphygmic interval and the transmission time vary inversely with the frequency of the ventricular contractions, and are relatively short when diastole is prolonged; auricular systole, even in cases of mitral stenosis, rarely lasts for more than 0.10 second; and when the *a-c* interval measures more than 0.2 second the conduction of the stimulus to the ventricles is unduly delayed.

The measurement of the *a-c* interval is apt to be fallacious

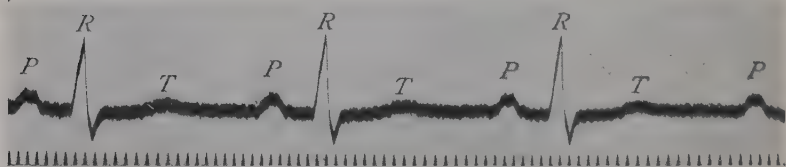


FIG. 103.—DEPRESSION OF CONDUCTIVITY.

The *P-R* interval is prolonged (0.21 second). From a man aged forty-one, suffering from aortic and mitral incompetence. Derivation *II*.

if the commencement of the *a* wave is not clearly defined. For example, when the heart is beating frequently the period of ventricular contraction may overlap the period of the succeeding auricular contraction, and the *a* wave may then be merged in the preceding *v* wave (Fig. 64). A similar overlapping of the waves *h* and *a* may occur. In such cases the auriculo-ventricular interval should, if possible, be determined from an apical tracing which will show that the *a* wave does not begin until after the start of the composite *v* or *h* wave of the jugular tracing.

In mitral stenosis a prolonged auriculo-ventricular interval may very rarely be recognized by the occurrence of a murmur which is mid-diastolic in time, instead of being presystolic. The murmur not only begins but also ends an appreciable

time before the first sound, because the auricular precedes the ventricular contraction by an interval somewhat longer than normal. Still more rarely, when the auriculo-ventricular interval is so prolonged that the ventricular response is delayed until the auricles are again in contraction, no presystolic murmur is audible, the first sound at the apex has a snapping character, and the only obstructive murmur of mitral origin is an early diastolic murmur.¹ Fig. 104, illustrating this depression of conductivity, was obtained from a miner aged thirty who had complained of shortness of breath for four years, but who had not suffered from dropsy. In this figure the *P-R* interval is 0.28-0.31 second, nearly

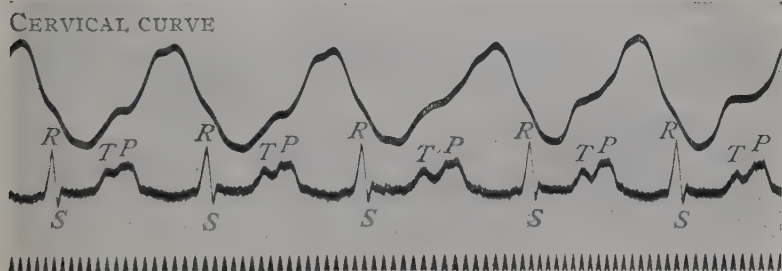


FIG. 104.—DEPRESSION OF CONDUCTIVITY.

The *P-R* interval is prolonged (0.28 to 0.31 second), *P* beginning before the antecedent *T* is completed. From a case of mitral stenosis by Derivation II.

twice its normal length, and *P* begins before the end of *T*. In the upper, cervical, curve of Fig. 104 each of the waves *a* is fused with the *v* wave of the preceding beat. The most common cause of disappearance of a presystolic murmur is, however, the onset of auricular fibrillation (see Chapter XI).

Depression of conductivity is not often observed and never causes any symptoms, although the patients in whom this defect is found have usually obvious symptoms and signs of disease of the myocardium, with or without valvular disease. The latent defect may be intensified or revealed by the vagus stimulation resulting from the administration of digitalis or of strophanthus. The defect may also be revealed by

¹ Ritchie, W. T., *Edin. Med. Journ.*, 1912, N.S., x., 410.

digital compression of the vagus in the neck. Each time this nerve is firmly, but momentarily, compressed the pulse "intermits" a beat, and synchronously there is silence over the præcordia.

(2) Partial Auriculo-Ventricular Block

This is a more severe grade of defective conductivity, for there is not merely delay in the transmission of stimuli to the ventricles but an occasional stimulus fails to reach them (Fig. 105). In other instances every second, third or fourth

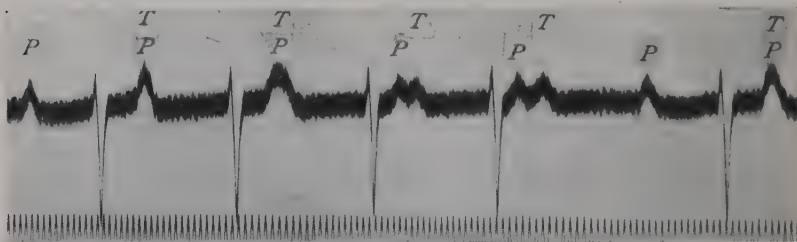


FIG. 105.—PARTIAL AURICULO-VENTRICULAR BLOCK.

The first *P-R* interval is prolonged (0.40 second), and successive *P-R* intervals become still longer, *P* coinciding with or preceding *T*, until the fifth auricular deflexion, *P*, is not followed by a ventricular complex. The fifth stimulus is blocked. The next ventricular complex begins 0.40 second after the sixth auricular deflexion, *P*. From a man aged sixty-one, suffering from aortic incompetence, tabes dorsalis and diabetes mellitus. Derivation *III*.

stimulus is blocked, and the condition is then termed 2 to 1, 3 to 2, or 4 to 3 block.

With each contraction of the heart all its inherent functions are in abeyance for the time, the stimulus material being exploded, the excitability lost, contractility and conductivity abolished. In health all these functions are rapidly restored during diastole. When conductivity is defective, its restoration after each systole is slower than normal, and the frequent exercise of the function entails further defect. In the upper record of Fig. 106, from a patient suffering from rheumatic fever, conductivity is so defective that even after the ventricles have rested for 1.65 second the *a-c* interval measures 0.30 second. The succeeding auricular contraction

occurs 0.85 second later, and the shorter rest necessitates a longer period (0.40 second) for the transmission of the stimulus; and when another auricular contraction occurs 0.75 second later, the stimulus fails to pass, and a ventricular contraction is missed. The longer rest which thus ensues improves conductivity, and ventricular contraction succeeds the ensuing auricular contraction by a shorter interval, and the process is repeated indefinitely. The radial pulse may be almost regular in these cases if every second ventricular

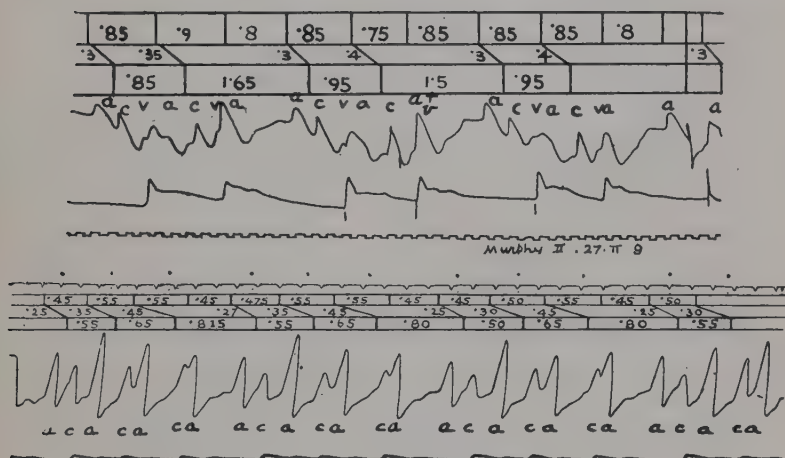


FIG. 106.—PARTIAL AURICULO-VENTRICULAR BLOCK.

In the upper record every third ventricular contraction fails. In the lower record every fourth ventricular contraction fails. The cardiac mechanism is shown in the diagrams above the cervical curves.

contraction fails; or every third or fourth ventricular beat alone may be missed.

If the heart is maintaining its sinus rhythm, partial block is always a sign of organic disease of the bundle. It may occur at any age in patients suffering from rheumatic fever, diphtheria, or any other acute infective disease, in whom there is probably an acute myocarditis involving the bundle. Transient defects of conductivity are not uncommon in acute rheumatic endocarditis, and we have observed eleven examples. In one case the defect persisted for nearly four weeks; in another it disappeared after six days, recurred

three days later, and again lasted for six days. In none of these cases was there complete block, but in one the ventricles only responded to every second sinus stimulus. All the cases made good recoveries, and in one alone was the ultimate valvular lesion severe.

Partial auriculo-ventricular block is, however, more often observed in elderly patients affected with chronic myocarditis, in the course of which the bundle has become fibrous.

Cases with partial block have no necessary inconvenience from their disability. In some instances the block may become complete for a time, the pulse may run at a very slow rate, and the Adams-Stokes syndrome may ensue (*see* p. 127). But in the majority of cases the defect is recognized on critical examination of an irregular pulse or on routine examination of a failing heart. Symptoms of cardiac insufficiency may or may not be apparent, but if present they are only in small degree the result of the block. Palpitation is frequently experienced by these patients. Partial block is usually aggravated by digitalis, and may be lessened temporarily by atropine.

The auricular rhythm in partial block may be somewhat inconstant. This is shown in Fig. 107. In the upper part of the figure the sphygmie periods are plotted out, and it is evident that the shorter inter-auricular periods of 0.55 and 0.65 second succeed synchronous contraction of auricle and of ventricle; the periods being shorter, the more nearly do the commencements of auricular and ventricular contraction coincide. To *a4*, occurring immediately *after* a ventricular contraction, succeeds an *a-a* period of 0.70 second; while to *a5*, which occurs in the middle of ventricular contraction, succeeds a period of 0.55 second. *a6* occurs during ventricular diastole and is followed by an auricular contraction after an interval of 0.70 second. When the auricles and ventricles contracted synchronously, the former were unable to expel their contained blood into the latter; the great veins became overfilled; and when the auricles passed into diastole the sudden rise of pressure within them from the venous inflow induced a premature contraction.

When there is a pronounced increase in the frequency of the stimuli which the bundle has to transmit to the ventricles,

partial block is liable to occur, despite the lesion in the bundle being comparatively slight. Consequently partial block may be observed in auricular flutter, in which disorder the auricular rate may be 320 per minute while the ventricular rate may be only 80 (*see* p. 168). Further, in auricular fibrillation an infrequent ventricular rate is a sign of defective auriculo-ventricular conductivity. The defect may be organic from fibrosis of the bundle, or functional as a result of the administration of digitalis or strophanthus. Indeed in auricular fibrillation the benefit resulting from these drugs is mainly, if not wholly, due to their depressing the conductivity of the bundle, thus lessening the number of stimuli which it

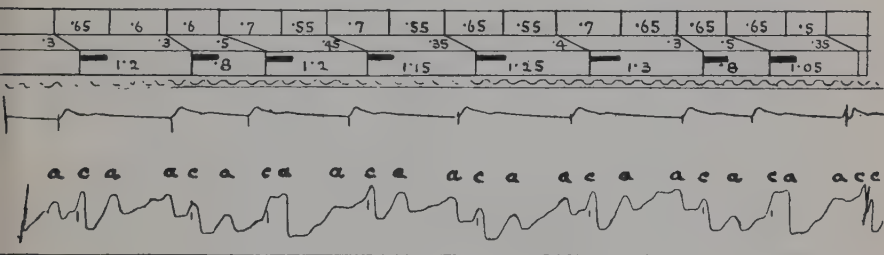


FIG. 107.—PARTIAL AURICULO-VENTRICULAR BLOCK.

Radial and jugulo-carotid tracings, with diagram of the rhythm of auricles and ventricles. The time-marker below this diagram records tenths of a second. The auricular contractions succeeding synchronous contractions of auricle and ventricle are premature.

transmits to the ventricles, and thereby reducing the ventricular rate (*see* p. 379).

(3) Complete Auriculo-Ventricular Block

In this condition, which is usually spoken of as complete heart-block, the auricular and the ventricular contractions occur independently of one another. There is complete auriculo-ventricular dissociation. The auricular contractions are usually normal in their rate and rhythm, and they are in response to sinus stimuli because the auricular rate alters on exertion, etc., as in normal hearts. But the ventricular contractions are infrequent, numbering about 30 to 36 per minute, are usually rhythmic, and are the result

of impulses arising in the ventricular portion of the bundle below the point at which it is severed.

The disorder is not common. We have observed twenty-three cases ; with one exception all were males, the youngest

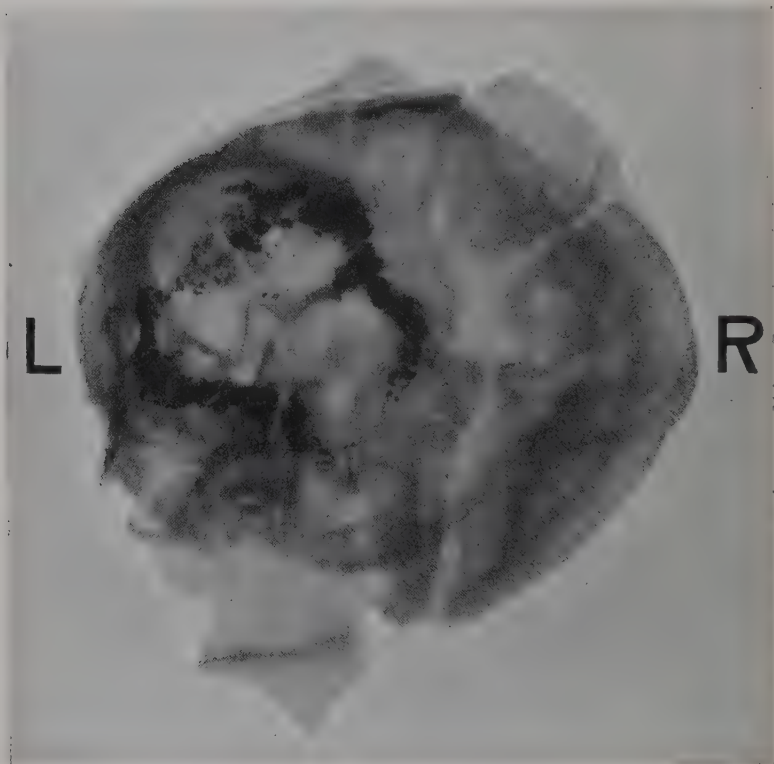


FIG. 108.—RADIOGRAM OF HEART, IN THE PLANE OF THE MITRAL ORIFICE, SHOWING A CALCAREOUS RING IN THE VENTRICULAR MUSCLE NEAR THE MITRAL ORIFICE, AND INVOLVING THE MEMBRANOUS SEPTUM. THE CALCAREOUS RING SEVERED THE BUNDLE AND CAUSED COMPLETE AURICULO-VENTRICULAR BLOCK.

MALE AGED SIXTY-FOUR. $\left(\times \frac{4}{5.5}\right)$

being aged thirty-three, the eldest seventy-nine. The lesions which have been found in the auriculo-ventricular node, the bundle and its branches are of various kinds. In some they are of acute character—abscess, arterial occlusion, etc.—in others they are chronic—a dystrophic fibrosis, tumour, gumma,

etc.—and acute or chronic inflammatory reactions may occur in their vicinity. Complete block is occasionally found in association with congenital patency of the ventricular septum. The following list of lesions found on *post-mortem* examination to be involving the auriculo-ventricular node or bundle is derived from those of Hirschfelder and Lewis: Gumma, 7; calcification, 7; fibrosis, 11; tumour, 2; anæmic infarct, 2; round-celled infiltration, 4; mural ulceration, 1; fatty degeneration, 1; arterio-sclerosis of the auriculo-ventricular artery, 1. If the lesion severs the bundle or both its main branches complete and permanent auriculo-ventricular block results; and conversely persistent complete auriculo-ventricular block signifies a lesion severing the bundle or both its main branches. In every well-authenticated case of persistent complete block, microscopic examination has demonstrated gross disease either of the bundle or of both its main branches. In the few cases where the continuity of the bundle appeared to be intact there was either no proof of complete block, or the microscopic examination was incomplete. We have examined seven cases of complete auriculo-ventricular block. In six, the main stem of the bundle was severed by chronic inflammatory, and in some instances by calcareous, lesions; in the seventh case, one investigated by Miss Meiklejohn, the node and bundle were intact, but both the main branches were almost wholly obliterated by dense fibrous tissue of inflammatory origin.

Before the lesion finally severs the bundle it may irritate and provoke a somewhat frequent ventricular rate. When the bundle is extensively diseased, yet not completely severed, the conduction of stimuli may be interrupted wholly, from time to time, by direct or by reflex vagal stimulation. The ventricles then stand still for a period lasting for several seconds (Figs. 114, 115, 116), and thereafter contract at a slow (15 to 30 per minute) and often irregular rate. It is then that we may observe the phenomenon of an “ascending staircase” in which the ventricular beats become successively stronger (Fig. 114). Moreover, fresh lesions, or an exacerbation of the secondary inflammatory reaction, may repeat from time to time and for an indefinite period the symptoms which ensued at the onset. An old scar is unirritating, and

the chronic form of auriculo-ventricular block with a regular, infrequent pulse is its manifestation.

The majority of these cases are thus chronic in their nature, but auriculo-ventricular block may occur in acute disease. In 1905 Mackenzie reported the transient occurrence of the milder grades of block in patients who were suffering from acute rheumatism and influenza, and since then several cases of complete heart-block, with *post-mortem* examinations, have been recorded (gonococcal endocarditis, ulcerative endocarditis, diphtheria, and acute rheumatism quickly followed by enteric fever). Clinically it has been observed, either complete or partial, in pneumonia, scarlatina and enteric fever.

Symptoms.—The chronic forms of complete block are not necessarily associated with any definite signs of cardiac distress, and Sir William Gairdner,¹ for example, who died at the age of eighty-two, enjoyed fair health for the last four years of his life, though there was complete auriculo-ventricular dissociation for all this period. In the majority of cases, however, some degree of cardiac insufficiency is present—weakness, shortness of breath upon exertion, etc.—though these are related to the causes which produced the block rather than to the block itself. The heart itself is rarely normal, for valvular flaws are common, and the musculature is apt to be affected diffusely by lesions similar to those which have involved the bundle.

The heart is usually enlarged and the apex-impulse forcible, and in many cases a mitral systolic murmur is audible.

The pulse is uniformly infrequent, numbering about 32 per minute, and is generally regular in rhythm. The rate may vary slightly from time to time, but it is little influenced by emotion, exercise, fever, etc., which produce notable effects upon a normal heart. The pulse is of larger volume, the systolic pressure is usually higher and the diastolic pressure often lower than in health.

Close inspection of the jugular pulsations may furnish evidence of the auricles contracting at a rate which is faster

¹ Gibson, G. A., and Ritchie, W. T., *Edin. Med. Journ.*, 1909, N.S., ii., 315, 507.

than that of the ventricles. This was described by Stokes¹ in 1854 as "a very remarkable pulsation in the right jugular vein . . . most evident when the patient was lying down. The number of the reflex pulsations is difficult to be established, but they are more than double the number of the manifest ventricular contractions. About every third pulsation is very strong and sudden."

Auscultation furnishes another sign. If the pulse is regular and a mitral systolic murmur co-exists, regurgitation into the auricle will cease when ventricular and auricular contraction coincide, and consequently the murmur will disappear or weaken.

In the vast majority of cases auricular contraction does not produce an audible sound. Over the precordia, however, a faint sharp sound due to auricular contraction may sometimes be heard. In only two of our cases was it clearly audible. The auricular sound is synchronous with an auricular wave in the jugular vein, and like the latter occurs at a varying moment during diastole. The time relation of the auricular sound to the second cardiac sound is therefore variable.

The complete dissociation of the auricular contractions from those of the ventricles is usually distinct in cervical, and often in apical, tracings (Fig. 109). The former reveal no regular relation between the *a* and the *c* waves, *a* now preceding, now following, and now coinciding with the carotid beat. When the *a* and *c* waves coincide the former is large, because the auricles are contracting at the same time as the ventricles, the auriculo-ventricular valves are shut, and the auricular blood is therefore thrown backwards into the great veins. In an apical tracing the *a* wave is generally lost when auricular and ventricular systole coincide, but is distinct during ventricular diastole (Fig. 109).

In electrocardiograms, complete dissociation is easily recognized. Fig. 110 was obtained from a man aged seventy-five, who had previously presented the Adams-Stokes syndrome. The auricular deflexions, *P*, occur rhythmically at a rate of 97 per minute, the ventricular deflexions are rhythmic at a rate of 38 per minute. There is complete auriculo-

¹ Stokes, W., *Diseases of the Heart*, 1854, 315. *Dublin Quart. Journ. of Med. Sci.*, 1846, ii., 73.

ventricular dissociation, but the form of each ventricular complex indicates that the ventricles are contracting in response to stimuli which reach them by the normal paths.

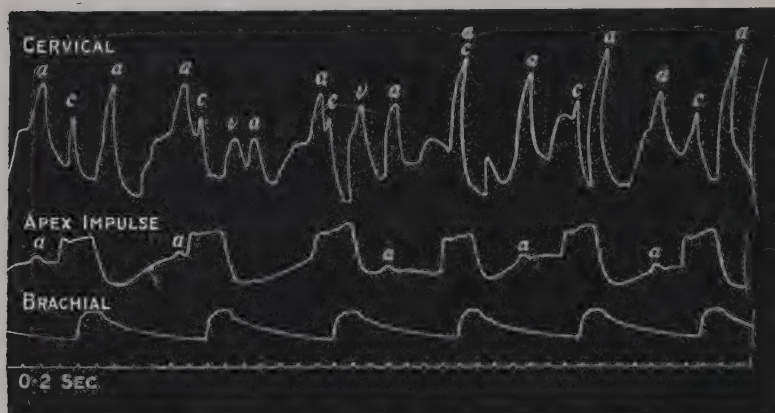


FIG. 109.—SIMULTANEOUS TRACINGS FROM A CASE OF COMPLETE AURICULO-VENTRICULAR BLOCK.

The auricles are contracting rhythmically at a rate of 78.2 per minute; the ventricles are contracting rhythmically at a rate of 42.8 per minute.

In some cases of complete block, the duration of ventricular systole, as determined by the *R-T* interval, is prolonged, lasting 0.49 to 0.56 second, as contrasted with the normal,

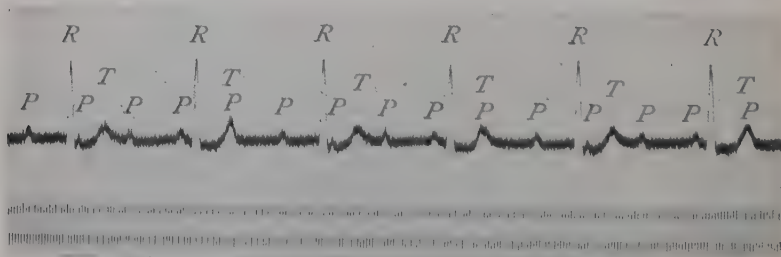


FIG. 110.—COMPLETE AURICULO-VENTRICULAR BLOCK.

The auricular rate is 97, the ventricular rate is 38, per minute. Derivation *I*.

which is about 0.30 second. In Fig. 111, from a case of complete block of the bundle and of its right branch, the *R-T* period is 0.66 second.

When the heart is examined radioscopically the auricular pulsations are seldom sufficiently obvious to justify, by this means alone, a diagnosis of auriculo-ventricular block; but in some cases the relative rapidity of the auricular contractions and their complete dissociation from those of the ventricles are easily recognized.

In complete block, ventricular extrasystoles are either interpolated, or are followed by a pause which is equal to that after the rhythmic ventricular beats.

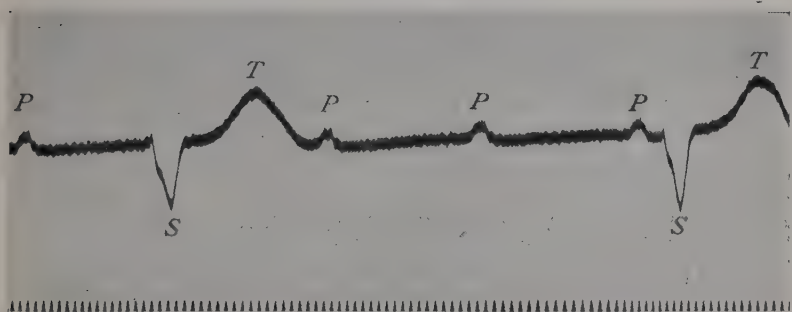


FIG. 111.—COMPLETE AURICULO-VENTRICULAR BLOCK AND BLOCK OF THE RIGHT BRANCH OF THE BUNDLE.

The duration of ventricular systole is 0.66 second. Derivation III.

THE ADAMS-STOKES SYNDROME

In auriculo-ventricular block a very striking series of symptoms, the Adams-Stokes syndrome, is not infrequently observed. The first case, recorded by Morgagni¹ in 1762, was followed by that communicated by Spens of Edinburgh to Andrew Duncan,² by that of Adams³ in 1827, and of Stokes⁴ in 1846. The syndrome is characterized by syncopal or epileptiform seizures due to transient standstill of the ventricles. The following is a typical example :—

The patient, an engineer, aged fifty-seven, had been employed in tropical climates for some thirty years. His health had been good

¹ Morgagni, *De Sedibus et Causis Morborum*, Venet., MDCCLXII, Lib. i., Epist. ix., Art. 7.

² Duncan, A., *Medical Commentaries for the Year MDCCCXII*, Edin., 1793, 458.

³ Adams, R., *Dublin Hosp. Rep.*, 1827, iv., 396.

⁴ Stokes, W., *Dublin Quart. Journ. Med. Sci.*, 1846, ii., 73.

while abroad, save for occasional attacks of ague. He had suffered from gonorrhœa twenty years before, accompanied by a sore which was not considered to be syphilitic, and was not followed by any secondary symptoms. Four years before his illness, however, he began to "feel the tropics," and two years later he came home on a year's furlough. He did not return to his work, as he felt that he was not fit for it, though he had no definite symptoms save on one occasion slight swelling of the feet, which disappeared in a few days.

The syndrome commenced suddenly three months before he came under our observation. He was going to a race-meeting, had been late in rising, and his breakfast had been hurried. While walking smartly to the railway-station, he suddenly felt faint and had to clutch hold of some railings to prevent himself from falling; but the faintness passed away in a few moments. When walking to the luncheon-stand in the afternoon he again felt faint, and on this occasion lost consciousness and fell to the ground, but he rapidly recovered. After lunch he felt better and stayed on at the races. He slept well at night, but next morning, while sitting quietly in his rooms, he had some thirty seizures of a similar kind.

These attacks recurred at irregular intervals. He was sometimes free from them for as long as a week, but sometimes they recurred several days in succession. On one occasion he fell down and cut his face. Two weeks before admission the attacks became more numerous and more severe. For hours at a time, they recurred frequently, and lasted for longer periods, and were associated with twitchings of the muscles, and in consequence he felt weak and exhausted. They were generally preceded by a dull pain in the upper part of the right chest.

For three days after his admission to hospital the attacks did not recur, but they recommenced next day at 7 a.m., and continued all day at short intervals; some 200 occurred in the twenty-four hours. Next day he had seven attacks, and on the succeeding day thirteen, but there were only eleven in the next eight days, and about twenty on the last day of his residence. They subsequently continued to be infrequent and of short duration.

Many attacks were observed. They were all similar in type. The first thing that was noticed was the cessation of the apex impulse and of the radial pulse. Simultaneously he said that he felt giddy and that a fit was impending, his countenance became white and anxious, and he moved his arms restlessly and tried to catch hold of the bedstead. In two or three seconds he became unconscious, and a slight tonic spasm, which sometimes seemed purposeful, ensued; and this was followed, if the ventricular asystole continued, by clonic spasms, which commenced in the face and spread to the arms and sometimes also to the legs. In a few seconds the face suddenly flushed, and the apex impulse and radial pulse reappeared; he straightened his head, and consciousness returned.

Convulsions did not occur in every attack. Many were of very short duration, and only lasted for five or six seconds (Fig. 115), in which case

no convulsion ensued, but others lasted for ten to fifteen seconds, and were then accompanied by muscular movements. These, however, even when general, were never gross, and incontinence never occurred. Recovery after the attacks was rapid and complete, but he said that he felt tired and exhausted.

The arteries were hard and tortuous, and the blood-pressure measured 110 to 145 mm. Hg. The pulse as a rule was fairly regular, about 28 to 32 per minute (Fig. 113), while the auricular rate was about 66 per minute, and at these times attacks did not occur. On other occasions the pulse was extremely irregular (Fig. 115), and a pause of several seconds might appear between successive beats, or the pulse rate might increase, and number even 56 per minute, a rate which was observed on two or three occasions.

The attacks may be of varying nature and duration. The shorter seizures of a few seconds' duration are syncopal, whereas convulsions, both tonic and clonic, usually supervene in those lasting for 10 seconds or more (Fig. 116). Some patients have only a few short attacks within a period of a few weeks or months, and thereafter survive for some years in comparatively fair health before terminal heart failure ensues. One of our patients had a final syncopal attack at the age of sixty-six, and died five years later of cerebral hæmorrhage causing hemiplegia and aphasia. Another, at the age of fifty-five, had several attacks, in the last of which he fell from a ladder. After that event the block was complete, and the ventricular rate was almost constantly thirty-six per minute. It was nearly ten years later before he became confined to bed on account of heart failure from which he ultimately died. Another of our patients had a first seizure at the age of sixty-nine, and a second and final seizure six years later, when he fell, unconscious, and burned his arm against a gas stove. When seen a fortnight later the block was complete, the auricular rate being 88, and the ventricular 32 to 33 per minute. He died a month later. Although in some patients the attacks are isolated events they may recur after intervals of weeks or months, over a period of two years, as in the case of Sir William Gairdner,¹ or for fourteen years as in one case recorded by Mackenzie. In other patients attacks recur at short intervals for hours on end, or even for a day

¹ Gibson, G. A., *Life of Sir William Tennant Gairdner*, Glasgow, 1912, 138-151.

or two. One of our patients, aged seventy-four, had occasional short syncopal attacks for several months. In spite of atropine the attacks became more frequent, compelling the patient to spend most of the day lying on a couch. He had no pain or distress during the attacks, for each of them was attended by pleasant dreams of journeys to the country or to the sea-coast. A few days before his death the block was apparently complete, but the attacks recurred several times an hour. In the longer convulsive attacks, lasting for two minutes, the breathing was rapid, deep, and stertorous, resembling that during the dyspnoëic phase of Cheyne-Stokes breathing.

It must clearly be understood that heart-block and the Adams-Stokes syndrome, though frequently associated, are not synonymous. Many cases of complete auriculo-ventricular block without the syndrome have been recorded, and the syndrome may occur without block. In complete auriculo-ventricular block the pulse is regular and numbers about 32 per minute. In the syndrome the pulse is irregular and very infrequent, numbering 5 to 20 per minute, and long intervals of many seconds' length may occur between the ventricular beats. The latter feature is the essential point. Kussmaul and Tenner showed that cerebral anæmia, induced by ligature or compression of the carotid arteries, produced loss of consciousness, and, if continued, convulsion; and observations of many attacks by numerous writers have made it clear that the ventricular contractions cease prior to the onset of cerebral symptoms. Cessation of the ventricular contractions occurs when a pronounced partial block becomes, from time to time, complete. Each time this occurs the ventricles stand still until another sinus stimulus is transmitted to them, or until the dormant rhythm of the bundle is developed.

The Adams-Stokes syndrome is most often observed in cases of chronic auriculo-ventricular block; it very rarely occurs in association with acute inflammatory lesions of the bundle. We have seen one fatal case in the course of diphtheria. In W. D. Macfarlane's case of acute rheumatic endocarditis, the pulse rate fell as low as 15 per minute, with occasional long intervals between the beats. On these

occasions the patient lost consciousness, and the muscles of the face and arms twitched. The convulsions recurred for two or three weeks, but recovery ensued after a tedious convalescence. He lived for more than ten years, and eventually died from chronic valvular disease.

In very rare instances the Adams-Stokes syndrome recurs after auriculo-ventricular block is complete and permanent. These attacks are due either to the ventricles suddenly developing a very extreme speed of contraction owing to irritation of the bundle below the lesion which severed it, or to ventricular standstill for reasons which are still obscure. The standstill is not due to vagus inhibition, because the ventricles in complete auriculo-ventricular block are beyond vagus control.

Sir William Osler¹ reported two cases of the syndrome in which heart-block seems definitely excluded. In one the patient suffered from tubercular disease of the first and second cervical vertebræ, and had on several occasions attacks of syncope and slow pulse. Another patient suffered from a sarcoma of the medulla, and experienced similar seizures.

The Diagnosis of complete auriculo-ventricular block offers no difficulty. The regular, infrequent pulse at a rate of 30 to 36 per minute, and the constancy of its rate in spite of emotion, change of posture and physical exertion, are characteristic. Moreover, the ventricular rate is the same as that of the pulse, and inspection of the jugular pulsations may reveal not only the higher speed of the auricles but also the complete auriculo-ventricular dissociation. In very rare cases of complete block the pulse rate is between 40 and 50, but a tracing from the jugular veins will demonstrate the auriculo-ventricular dissociation. In *sinus bradycardia* the rate of the heart is seldom below 40, and it varies with breathing, posture and exertion (*see* p. 81). A markedly infrequent pulse rate is observed in some cases of *auricular fibrillation* particularly after large doses of digitalis have been administered, but the rhythm of the pulse is obviously irregular, and its rate seldom falls

¹ Osler, Sir Wm., Allbutt and Rolleston's *System of Medicine*, Lond., 1909, vi., 145.

below forty and often varies considerably from hour to hour, or from day to day. In complete block the pulse rate is slower and more constant than that in *nodal bradycardia*. In *coupled rhythm* the pulse rate is markedly infrequent when an extrasystole recurring after each physiological beat fails to open the aortic valve; but auscultation reveals the sounds of two ventricular beats for each pulse wave.

The Adams-Stokes syndrome is most likely to be mistaken for syncope, but the characteristic train of events can hardly be confused with those of epilepsy, Jacksonian epilepsy, apoplexy, or the seizures occurring in general paralysis of the insane.

The Prognosis in auriculo-ventricular block is based upon two factors, the degree of the block and its cause. In general the prognosis is grave, for the block, whether partial or complete, is an indication of serious myocardial disease. The presence of valvular lesions, their nature and severity, are also of some moment. The auriculo-ventricular node and bundle are not infrequently affected in valvular disease, but this is owing to their close proximity to the valves, and the myocarditis is not necessarily widespread. If the lesion is due to an acute process it may subside, as in acute rheumatism, or progress as in malignant endocarditis; if due to chronic arterial disease it will steadily increase. A syphilitic lesion may resolve under treatment (Osler). The presence or absence of signs of myocardial insufficiency is necessarily important.

A partial block may become complete or may pass away. The dangerous period is that when lesions are forming in the bundle, and particularly when there is a high grade of partial block. It is at this stage that there is a constant liability of the block becoming complete, with consequent standstill of the ventricles until further sinus stimuli can be transmitted to them or until stimuli are formed in the bundle below its point of severance. The experience of the individual patient is important; and a fall of the pulse rate below 30 per minute or the occurrence of even trivial syncope or convulsion renders the immediate prognosis extremely grave.

Even in the eighteenth century physicians could foretell a syncope attack by the increasing rarity of the pulse.¹

Chronic, complete heart-block is not necessarily incompatible with many years of fair health; when the block is fully established and the scar in the bundle is contracted and quiescent the danger of sudden death from ventricular standstill is almost invariably past.

Treatment.—In partial block complete rest is certainly indicated, for exertion is bound to increase the work of the heart, and thus to increase in some measure the inflammatory reaction and the ultimate scar. The administration of digitalis is contra-indicated, for it tends to exaggerate defective conductivity, and vagal compression has a similar effect. In certain cases the administration of atropine ($\frac{1}{100}$ to $\frac{1}{50}$ grain) has lessened or abolished the block and so increased the number of the ventricular contractions, and it should certainly be given if the ventricular rate becomes infrequent. In most cases it has no action of this kind. The diffusible stimulants, strychnine and hot fomentations to the præcordia, are most likely to be useful in patients with syncope.

In complete block, digitalis, atropine, and vagal compression have no influence upon the rate of the ventricular contractions.

BUNDLE-BRANCH BLOCK

After the experimental division of one or other main branch of the bundle, stimuli are transmitted to both ventricles by the intact branch alone. The form of the ventricular complexes then presents characteristic alterations;² and when comparable electrocardiographic records are obtained clinically the condition is termed bundle-branch block (Fig. 117).

The characteristic ventricular complex is diphasic, consisting of an initial deflexion, or *Q-R-S* group, which lasts longer than the normal 0.10 second, and of a terminal deflexion,

¹ Morgagni, *De Sedibus et Causis Morborum*, Venet., MDCCLXII, Lib. v., Epist. lxiv., Art. 5.

² Eppinger, H., and Rothberger, J., *Zeitschr. f. klin. Med.*, 1910, lxx., 1.

T, in the opposite direction. The initial deflexion is usually so prolonged that it occupies at least one-third of the whole period of ventricular systole. If the block is in the right main branch, each ventricular complex, of abnormal form as described above, resembles that of a left, apical, ventricular extrasystole, the initial deflexion being upward by Derivation *I*, and downward by Derivation *III* (Fig. 117). If the block is in the left main branch, the ventricular complex resembles that of a right, basal, ventricular extrasystole, the initial deflexion being downward by Derivation *I*, and upward

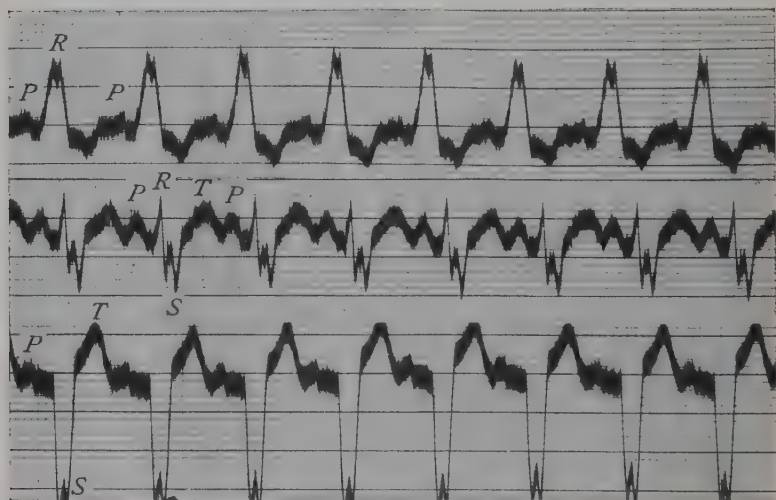


FIG. 117.—RIGHT BRANCH BLOCK IN A MAN AGED FIFTY-THREE.
DERIVATIONS *I*, *II* AND *III*.

by Derivation *III*. The block is more often in the right than in the left main branch, for the former is long and slender, whereas the latter spreads out fanwise and is therefore less liable to be severed.

This form of block is decidedly uncommon. It does not cause any characteristic symptoms, but may be detected in cases of myocarditis, with or without valvular disease. The recognition of this form of block depends wholly on the abnormal and distinctive form of the ventricular complexes. The records by Derivations *I*, *II*, and *III* in Fig. 117 are char-

acteristic of a block of the right main branch. The diphasic form of the ventricular complex is most conspicuous by Derivations *I* and *III*; the auricular deflexion, *P*, is largest by Derivation *II*.

The patient was a short, stout man fifty-three years of age, who had suffered from rheumatic fever at the age of thirty-seven, and who had been complaining of breathlessness on exertion and of palpitation, following bronchitis, for six months. The apex-impulse was in the sixth intercostal space, 15 cm. from the mid-sternal line; the rhythm was regular, the first sound was short and faint, the second accentuated and impure. The diastolic and systolic blood pressure were respectively 165 and 190 mm. Hg. He was not dropsical. Six weeks after the record was obtained, the patient resumed his work as a clerk, though still suffering from some discomfort on exertion. Eleven weeks

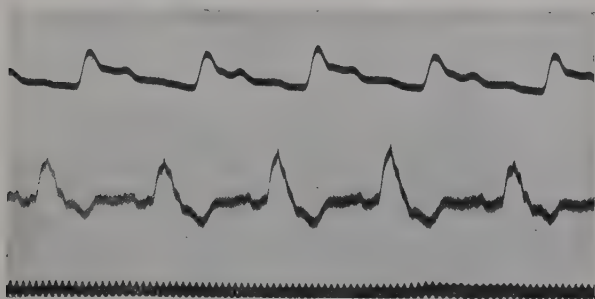


FIG. 118.—RIGHT BRANCH BLOCK.

Simultaneous record of brachial pulse and of electrocardiogram by Derivation *I*.

later he had a sudden attack of dyspnœa, with great irregularity of the heart, and died in a few hours.

Another patient was a drysalter, aged sixty-three, who had been suffering from dyspnœa for eighteen months. The heart was greatly enlarged, and the shadow of the aorta seen on the fluorescent screen was large and dense. The heart sounds were closed, but prolonged and muffled. The arteries were thick; transient alternation was observed after each extrasystole; the systolic blood pressure varied from 190 to 215 mm. Hg. and the urine contained albumin. The Wassermann test was negative. Three weeks after the records in Figs. 118 and 119 were taken, the dyspnœa became worse, and the patient lay propped up on six pillows. Dropsy set in and increased steadily in spite of digitalis, strophanthin intravenously, diuretics, Southey's tubes, and aspiration of the pleural sacs. Four months after his admission, cyclic breathing, drowsiness, vomiting and twitching of the hands and fingers

preceded the fatal termination. *Post-mortem* examination revealed cardiac dilatation and hypertrophy, slight atheroma of the aorta and coronary arteries, chronic nephritis, and venous congestion and cirrhosis of the liver.

The block may be organic and permanent, as in the two cases recorded above, but it may be functional and transient, as in the following case :—

The patient, a blacksmith aged forty-three, had suffered from chorea at the age of eleven, from rheumatic fever when twenty-three, and from several minor rheumatic attacks until he was twenty-nine.

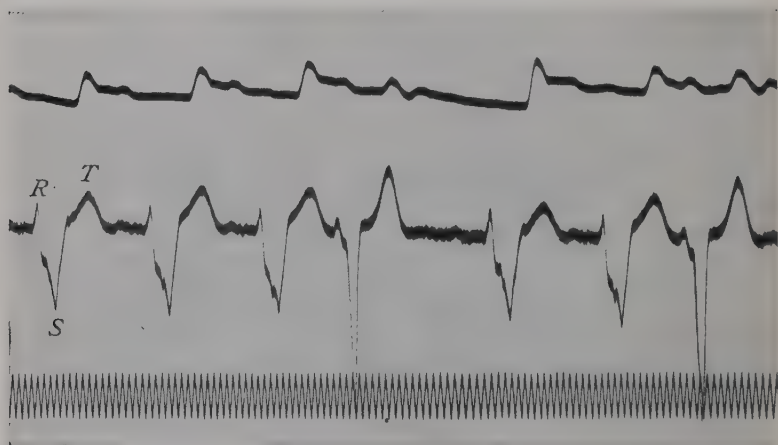


FIG. 119.—RIGHT BRANCH BLOCK.

Simultaneous record of brachial pulse and of electrocardiogram by Derivation *III*. Man aged sixty-three. The fourth and last beats are ventricular extrasystoles.

Thereafter he was well, and worked hard, until he became breathless fifteen months before his admission to hospital. He was then cyanosed, slightly jaundiced, and markedly breathless, but not dropsical. The heart was much enlarged, its borders being 6 cm. to the right and 10 cm. to the left of the mid-sternal line. A presystolic and an early diastolic murmur of mitral origin were audible. The systolic blood pressure was constantly about 170. The pulse, having a rate usually of 80 to 90 per minute, was alternating markedly, and after each extrasystole the alternate, smaller, waves could not be felt at the wrist (Fig. 128). The Wassermann reaction was negative. The urine was scanty at first, and though it never contained albumin, the phthalein excretion was only 26 per cent. in three hours. Each of the rhythmic ventricular contractions yielded a

complex almost identical with those in Fig. 117. The patient's progress was uninterrupted, except by one severe paroxysm of dyspnoea lasting for an hour and a half, during which the pulse was 140 per minute and markedly irregular. When he left hospital two months later, the block had disappeared.

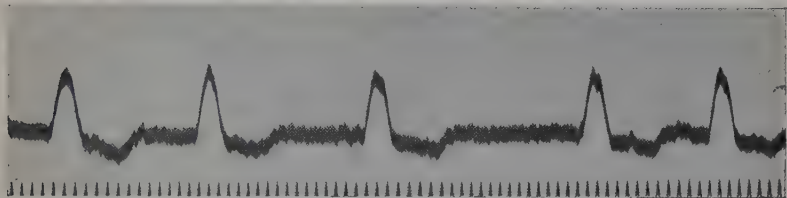


FIG. 120.—AURICULAR FIBRILLATION AND RIGHT BRANCH BLOCK.
DERIVATION I. Man aged fifty-five.

In a few cases the heart sounds have a canter, or gallop, rhythm, but this is exceptional. In rare instances branch block is induced by vagal stimulation. For example, when there is a slight, pre-existing defect of conductivity in the right branch, vagal stimulation may convert this defect

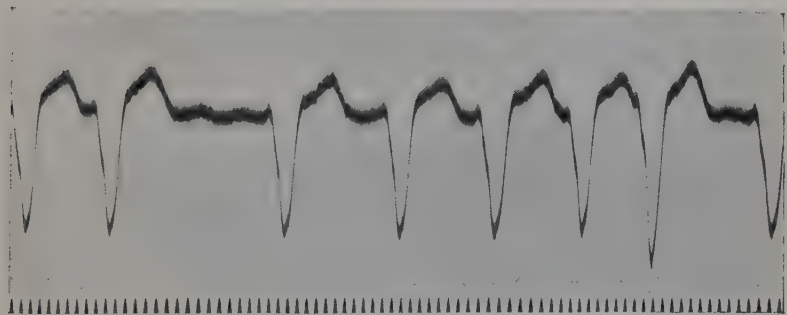


FIG. 121.—AURICULAR FIBRILLATION AND RIGHT BRANCH BLOCK.
DERIVATION III. Man aged fifty-five.

into a complete block, and the sinus stimuli are then transmitted only by the left branch.

If the continuity of both main branches is destroyed, complete auriculo-ventricular block ensues, as described on page 123. In other cases there is severance of the main stem of the bundle and also of its right branch, as in Fig.

111, which shows not only complete auriculo-ventricular dissociation but ventricular complexes characteristic of a right branch block. Each stimulus for contraction of the ventricles, originating in the bundle below the severing lesion, reached both ventricles through the left branch only.

The co-existence of bundle-branch block with auricular fibrillation, as illustrated in Figs. 120, 121, from a man aged fifty-five, is most exceptional.

In all the circumstances described above, branch block signifies myocarditis, which is usually chronic, widespread and severe. Occasionally, however, this form of block is observed in cases of tachycardia, owing to one or other main branch, but usually the right, suffering from fatigue by the frequent transmission of stimuli. A block developing under these circumstances disappears when the tachycardia ceases.

In cases diagnosed as bundle-branch block post-mortem examination may reveal a definite breach of continuity of a main branch, but the condition which is more often found is destruction of the lesser branches in the lower third of the ventricular septum by subendocardial fibrosis. This form of block consequently differs only in degree from that next to be described.

INTRAVENTRICULAR (ARBORIZATION) BLOCK

In this form there is defective conductivity in the terminal branches of the bundle. These branches form an arborization in the subendocardial layers of the ventricles, and are often extensively destroyed in chronic myocarditis. The patient usually presents signs of heart failure, with dyspnoea on exertion; there may be dropsy; angina is not common. The heart is enlarged, its sounds are muffled, the blood pressure is often high and the pulse may be alternating.

Figs. 122 and 123 were obtained from a labouring man, aged thirty-four, whose health was good until he became weak, breathless and giddy, three months before his admission to hospital. He never suffered from angina. A week before his admission the feet began to swell, and he became confined to bed. He had marked orthopnoea, slight cyanosis, and a moderate degree of dropsy. The apex-beat was in the sixth interspace, the sounds were feeble and gave at times a canter rhythm, but no valvular murmur was audible. The systolic

blood pressure was constantly over 200 mm. Hg. The sputum was abundant and muco-purulent; at the bases of the lungs many crepitations were audible. He died suddenly a fortnight after the records in Figs. 122 and 123 were obtained.

The characteristic features are electrocardiographic rather than clinical. The *Q-R-S* group of deflexions in each ventri-

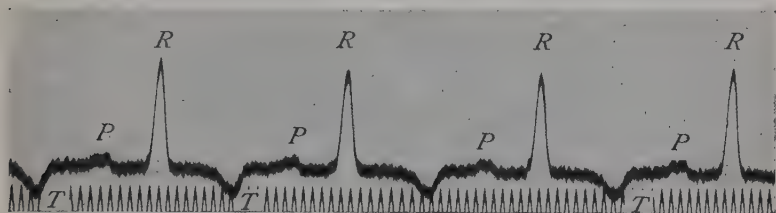


FIG. 122.—INTRAVENTRICULAR BLOCK. DERIVATION I.
Man aged thirty-four.

cular complex is prolonged beyond the normal 0.10 second,¹ the *R* deflexion is broad, and there is usually irregular notching or splintering of *R* or *S*, or of both (Fig. 123). The *Q-R-S* group of deflexions occurs while the ventricular muscle is passing into activity in response to a stimulus transmitted to it by the fine, terminal branches of the bundle. A delay

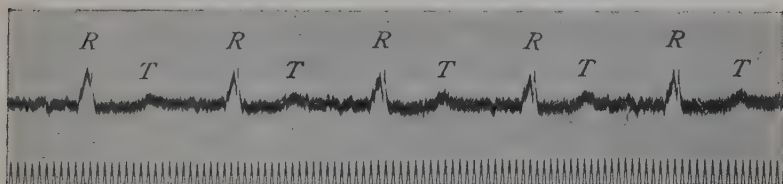


FIG. 123.—INTRAVENTRICULAR BLOCK. DERIVATION III.

in the transmission of the stimulus through the ventricles is shown by prolongation of the *Q-R-S* group; and an abnormal spread of the stimulus, whereby the different parts of the ventricular muscle are not activated in normal sequence, is shown by irregular notching or splintering of the *Q-R-S* group of deflexions. This conclusion is not endorsed by

¹ This may also occur in cases with left ventricular preponderance.

Wilson and Hermann,¹ who consider that the diagnosis of arborization block rests on a very insecure foundation.

Intraventricular block is not infrequent. Nearly all of our cases have been males, and with few exceptions they have been over forty years of age. All were suffering from obvious heart-failure. The prognosis is always grave. The least serious cases are those who develop intraventricular block in consequence of an excessive ventricular rate, whereby the terminal fibres of the bundle are not given sufficient time fully to regain their conductivity and consequently suffer, as indicated by Robinson,² from functional fatigue. Most of our patients presenting intraventricular block died within a few months; few survive for more than one year, or at most two years, after intraventricular block is detected.

SINO-AURICULAR BLOCK

Defective conductivity in the fibres connecting the sinus node with the right auricle is recognizable only when the defect amounts to a block. This is seldom observed,³ and attempts to produce sino-auricular block experimentally usually fail.⁴ The block is recognized by the whole heart missing a beat. Stimuli are arising rhythmically in the sinus node, but now and again a stimulus is not transmitted to the auricles and to the ventricles, and the whole heart therefore misses a beat. The succeeding stimulus is effective at the moment when it is due. The disorder is depicted in Fig. 124.

In rare instances, two, three, four or more sinus stimuli in succession fail to be transmitted; there is a prolonged diastolic arrest of the whole heart, and the patient may faint. The clinical condition is then that of the Adams-Stokes syndrome, but this is more frequently due to auriculo-ventricular, than to sino-auricular, block. Sino-auricular block has

¹ Wilson, F. N., and Hermann, G. R., *Arch. Intern. Med.*, 1920, xxvi., 153.

² Robinson, G. Canby, *ibid.*, 1919, xxiv., 442.

³ Wenckebach, K. F., *Arch. des maladies du cœur*, 1908, i., 65. Levine, S. A., *Arch. of Intern. Med.*, 1916, xvii., 153. Worth Brown, N., *ibid.*, 1919, xxiv., 458.

⁴ Eyster, J. A. E., and Meek, W. J., *ibid.*, 1917, xix., 117.

seldom been observed except in patients who were taking digitalis, and the disorder is mainly, if not entirely, a result of vagal stimulation. Because of the diffuse nature of the connections between the sinus node and the auricular muscle, this form of block is probably never due solely to organic disease.

The record in Fig. 124 was obtained from a man who was hale and active until the age of seventy-one when, stooping to pick up some papers, he fell down and was unconscious for a few minutes. A year later he suffered from two further syncopal attacks of short duration, and became breathless when walking. For the first time the pulse was then found to be irregular and infrequent, the rate being 28 to 38

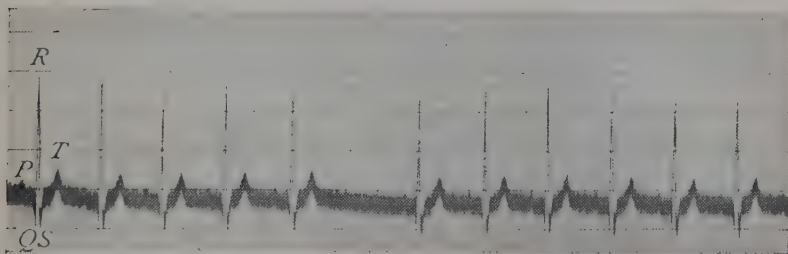


FIG. 124.—SINO-AURICULAR BLOCK. DERIVATION II.

The whole heart misses a beat.

per minute. Electrocardiograms revealed complete auriculo-ventricular block. The ventricular complexes were of normal type, but the *R-T* interval was prolonged to 0.48 second; the auricular deflexions were of normal form and were as infrequent as those of the ventricles. A week later the pulse rate rose to 36–48, and in the succeeding week to 60–72 per minute. Three months later he looked, and felt, well, the heart had a sinus rhythm, the auriculo-ventricular block had disappeared, but sino-auricular block was recorded (Fig. 124). In other records, two or three sinus stimuli in succession were blocked. One month later drowsiness and Cheyne-Stokes breathing developed, and he died in coma, evidence of sino-auricular block being obtained a few hours before death.

Although the sino-auricular block in this patient was associated with heart failure, this form of block is not necessarily of grave import, and may occur as a transient and functional defect in a heart which is otherwise healthy.

CHAPTER VIII

CONTRACTILITY

THE contractility of the ventricular muscle at the moment of contraction may be measured by the size, or volume, of the pulse wave, because the cardiac contraction is always maximal irrespective of the degree of the stimulus.

All the functions of the heart muscle, including its contractility, are abolished by each systole, but they are rapidly restored during diastole. If a contraction is premature contractility is necessarily less restored ; and as the antecedent diastole was short, the ventricle is imperfectly filled, while the aortic blood pressure is relatively high ; consequently a premature ventricular contraction causes a relatively small arterial pulse wave. Again, when the rate of the heart is accelerated, each diastole being shortened, the contractile force of each systole, and the volume of each pulse wave, are less than when the heart is beating more slowly. These variations in the contractility of the heart, depending essentially on the length of diastole, do not signify any impairment of contractility.

Defective Contractility

When the heart is failing the cardiac chambers usually become dilated and their muscle fibres, being stretched beyond their optimum length, contract at a mechanical disadvantage. The contractions are therefore enfeebled, the reserve force is lowered, and dyspnoea and dropsy often ensue. In cases of this nature both the tonicity and contractility of the heart muscle are probably lessened. But depression of tonicity does not necessarily imply defect of contractility ; and although all the clinical evidence suggests that when the heart is dilated beyond the physiological limit

its contractile force is lessened, this can seldom be demonstrated. There is, however, one clinical phenomenon, namely alternation of the pulse, which is usually regarded as evidence of defective contractility of the ventricles.

Alternation

An alternating pulse, or *pulsus alternans*, occurs when the ventricular contractions are rhythmic but alternately stronger and weaker. The pulse waves, too, are alternately larger and smaller. They may be regular, or the smaller waves may be slightly delayed, but they are never premature. A comparison of the pulse tracing with a simultaneous tracing from the apex impulse or an electrocardiogram shows that the delay of the smaller pulse wave is not due to delay in the onset of ventricular systole. Thus, in Fig. 131 the interval between the *R* of the third ventricular complex and the commencement of the brachial pulse wave is 0.21 second, whereas the corresponding interval of the next beat, which causes a

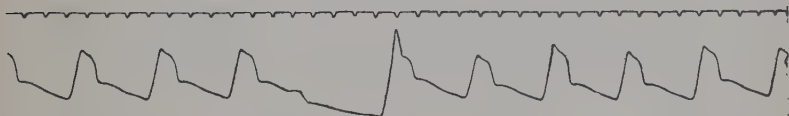


FIG. 125.

After three normal beats there is an extrasystole, and after this there is transient alternation of the pulse.

smaller pulse wave, is 0.24 second. The smaller pulse wave necessitates a longer presphygmic interval and transmission time.

Alternation may be continuous or transient. The latter

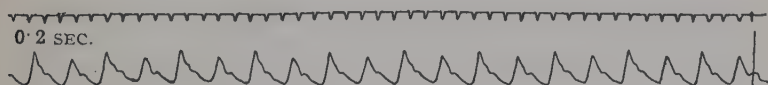


FIG. 126.—CONTINUOUS ALTERNATION. PULSE RATE 126 PER MINUTE.

From a case of mitral stenosis, aged fifty-three.

is the more common, the defect being latent until it is revealed in the pulse beats succeeding a transient irregularity such as an extrasystole (Figs. 125, 130). A continuous alternation is illustrated in Fig. 126, where the arterial pulse waves

are seen to be alternately larger and smaller, but rhythmic. Continuous alternation of varying degree is shown in Figs. 127, 129. When the alternation is very pronounced, the smaller waves may be imperceptible to the finger or in a tracing of the pulse. This is illustrated in Fig. 128, from a blacksmith aged forty-three, who was suffering from mitral



FIG. 127.

There is an extrasystole after the third beat, and thereafter the alternation becomes more pronounced.

stenosis and dilatation of the heart, and in whom there was a functional block of the right branch of the bundle. The heart had a sinus rhythm; the auricular beats were rhythmic, as shown by the cervical curve of Fig. 128; and the alternation is so pronounced in the second half of the brachial tracing that the smaller pulse waves are almost invisible.

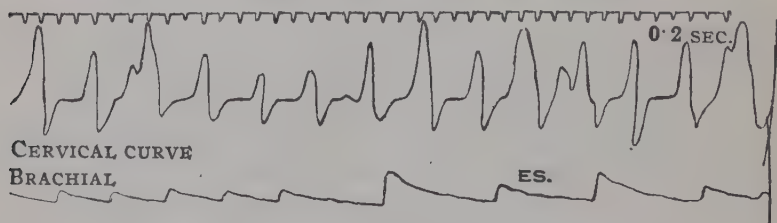


FIG. 128.—SEVERE GRADE OF ALTERNATION.

In the second half of the brachial curve the smaller pulse waves are almost invisible. Man aged forty-three, suffering from mitral stenosis and cardiac dilatation.

Transient alternation is seen in Fig. 125. The first three beats are practically of uniform size, and are succeeded by the small, premature, beat of an extrasystole. The fifth beat (post-extrasystolic) is larger than normal. The small size of the extrasystolic wave and the large size of the wave succeeding it are merely the sequel to the varying length of antecedent diastole. But the waves after the post-extrasys-

tolic beat are alternately smaller and larger, though the pulse periods are equal. Towards the end of the tracing the alternation becomes less evident.

The exact nature of the defect producing alternation is not yet fully understood. The hypothesis usually accepted is that of partial ventricular systole, namely the failure of some of the muscle fibres to contract in each alternate (weaker)

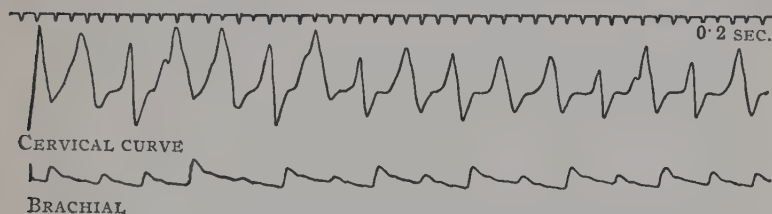


FIG. 129.—CONTINUOUS ALTERNATION OF VARYING DEGREE.

beat, because of defective contractility or of defective conductivity within the heart muscle, whereas a greater number of muscle fibres contract in the stronger beat.

On another hypothesis, the degree of diastolic filling of the ventricle is alternately larger and smaller because the antecedent systole is alternately shorter and longer. This may

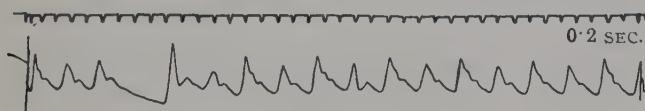


FIG. 130.

There is an extrasystole after the third beat, and subsequently the alternation of the pulse is evident.

be the reason why alternation, which is not otherwise noticeable, is often revealed in the first few beats after an extrasystole. Succeeding the extrasystole diastole is prolonged, the post-extrasystolic contraction is larger and more prolonged than usual and the diastole following it is therefore necessarily curtailed by the next ventricular systole occurring at the moment when it is due. The ventricular contractility, if defective, recovers imperfectly during the shorter diastole, and the ensuing systole is therefore less powerful and shorter. The shortness of systole entails prolongation of diastole, and

the next beat is therefore larger and longer ; and the process may be repeated indefinitely. The alternation, however, usually passes away gradually, to recur after the next extrasystole.

Alternation is one of the rarer forms of abnormality of the pulse, but it is more common than is often supposed. Minor degrees of alternation usually escape recognition when the pulse is felt by the finger, but are easily detected in pulse tracings. The disorder is most often found in patients past middle life, who are suffering from chronic myocarditis or myocardial degeneration as a late sequel either of sclerosis of the coronary arteries or, less often, of chronic valvular disease, while the heart maintains its sinus rhythm.

The patient usually complains of dyspnoea on exertion, more rarely of angina, and is unfit for work.

These were the chief symptoms in a patient who had suffered from rheumatic fever in youth, and who had taken alcohol to excess and acquired syphilis when a young man. At the age of fifty-three his health began to fail, and twice he fainted while at his work. The heart, somewhat enlarged, had a sinus rhythm with a rate of 126 per minute. The heart sounds had a canter rhythm, but no valvular murmurs were audible. Slight, continuous alternation of the pulse, though imperceptible to the finger, was revealed in a tracing (Fig. 126), and became well marked after each extrasystole, all of which were of ventricular origin (Fig. 130). Within a month he became wholly confined to bed, and lay propped up on the pillows ; the face acquired a tint indicating both cyanosis and slight jaundice ; he developed tenderness over the congested, enlarged liver ; the urine became scanty and albuminous in spite of diuretics. Eventually the heart became more dilated, dropsy which was never lessened by digitalis became steadily progressive, and he died, fifteen months after he gave up his work.

Another illustrative case is that of a lady aged seventy, who had been complaining of dyspnoea on exertion, and also of occasional præcordial pain and nocturnal paroxysms of dyspnoea for a year, and whose feet were slightly dropsical. The heart was not much enlarged, its sounds were pure, the pulse was beating rhythmically 118 times per minute, and alternation could be felt especially after extrasystoles, but these were not frequent. Relief from all symptoms was obtained by means of rest, dieting and digitalis. Three months later the patient was able to be up all day, and went freely about the house and garden ; the pulse was regular and no longer alternating. But nine months later, dyspnoea and dropsy recurred ; the pulse was rhythmic at a rate of 105, and again alternating markedly ; the sys-

tolic blood pressure was 195; the urine contained much albumin and many granular and hyaline casts. Death supervened within a week, two years after the onset of heart failure and one year after the alternating pulse was first observed.

In other patients the clinical features are primarily those of chronic nephritis, as in a man aged fifty-six who had albuminuria and whose systolic blood pressure was 217. His pulse, having a rate of 94 per minute, presented a slight and continuous alternation.

Alternation after an extrasystole may be observed in other cases, and particularly in elderly people who have arterio-

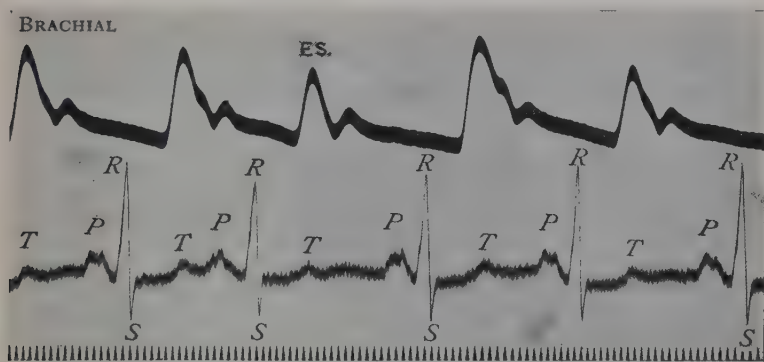


FIG. 131.—SIMULTANEOUS RECORD OF THE BRACHIAL PULSATIONS AND ELECTROCARDIOGRAM BY DERIVATION II, SHOWING ALTERNATION AFTER AN EXTRASYSTOLE (ES).

sclerosis but no very obvious cardiac symptoms. This was illustrated in a man, aged sixty-eight, suffering from fibroid phthisis and pulmonary emphysema with occasional hæmoptysis. The arteries were thick and tortuous; the systolic blood pressure was 140, the heart was beating with a sinus rhythm at a rate of 90 per minute. He presented an occasional extrasystole, and after each there was transient alternation.

If alternation be suspected, a pulse tracing should, if possible, be taken, for this may reveal an alternation which is imperceptible to the finger. If the patient is lying quietly in bed, alternation is apt to be missed. If he is able to undertake a little exertion, a latent alternation may be revealed, because the disorder is always more obvious when the cardiac

rate is accelerated, and tends to disappear when the rate lessens. This was illustrated in a man aged fifty-five, suffering from aortic and mitral incompetence; the heart had a sinus rhythm, but was failing; the cardiac rate was 103 and the pulse was alternating. Rest in bed and the administration of digitalis lowered the rate to 88, and the alternation then disappeared. That acceleration of rate alone may reveal a latent alternation is shown by its development after an injection of atropine in some cases of heart failure. Thus, an old man of seventy-six, suffering from arterio-sclerosis, presented an alternating pulse when the cardiac rate rose from 85 to 103 after $\frac{1}{40}$ grain of atropine.

While alternation is most often observed in cases of chronic myocarditis, it also occurs in pronounced tachycardia, and especially in auricular flutter, when the pulse rate reaches or exceeds 140 per minute. Moreover, it may be noticed as a rare and transient event during convalescence from pneumonia or other acute infective disease, or during chloroform anæsthesia. Although vagal inhibition is stated to depress contractility in animal experiments, it seldom induces alternation in man. After the heart's escape from the inhibition of vagus compression the pulse beats are usually of full amplitude and equal. In exceptional cases alternation ensues when the auricular contractions are synchronous with those of the ventricles.

Diagnosis.—Because of its grave prognostic significance it is most important to recognize alternation. It should always be suspected in patients who present symptoms of heart failure, however slight, whose arteries are thick, whose blood pressure is high, and whose pulse is regular in rhythm and not unduly fast. In cardiorenal cases alternation is not uncommon. We should always be on the watch for alternation after an extrasystole in patients who are past middle life, and remember that a pulse-tracing may reveal alternation which is not perceptible to the finger. Latent alternation may be revealed by physical exertion, and if this be impossible owing to the patient's condition, the application of pressure upon the artery distal to the sphygmograph may elicit it. Moreover, alternation may be detected when the blood

pressure is being ascertained by the auscultatory method. When the pressure within the armlet has been raised to the level of the systolic pressure, only every alternate, systolic, arterial sound is audible; and not until the pressure within the armlet has been lowered about 10 or 15 mm. Hg. is every systolic arterial sound audible. For example, in a lady aged sixty the arterial pressure rose to 240 and to 255 mm. Hg. in alternate beats.

The alternating pulse must not be confounded with extrasystoles. The distinction is easy, for an extrasystolic beat is not only small but *premature*, and is never delayed. The

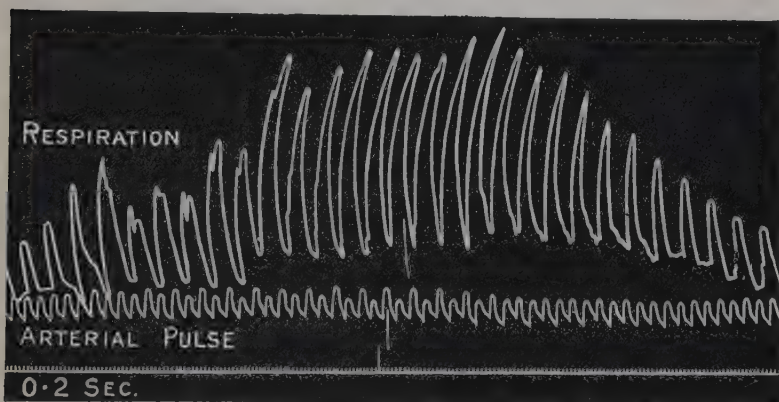


FIG. 132.—CHEYNE-STOKES RESPIRATION, WITH PULSUS PARADOXUS DURING THE PHASE OF DYSPNŒA.

alternating pulse may, however, be simulated closely during the dyspnœic phase of Cheyne-Stokes breathing (Fig. 132). In the central portion of this tracing the arterial pulse waves alternate in size, being smaller during inspiration than during expiration. This abnormality, a transient *pulsus paradoxus*, is of respiratory origin, and occurs during the dyspnœic phase when the respirations increase in frequency to such a degree that they number half the pulse rate.

In *Electrocardiograms*, T_1 may be small or inverted, the form of the ventricular complex may be that of left-sided preponderance or the *P-R* interval may be prolonged, but the records present no features characteristic of alternation.

Prognosis.—The *pulsus alternans*, when continuous, is always of evil omen, and its presence even if only for a few beats after an extrasystole is also serious. The only exceptions to this rule are the alternation due to obvious and remediable toxic conditions and that occurring in association with extreme grades of tachycardia. But in all other cases the prognosis is grave, and few patients survive for more than one or two years after the alternation is first observed. In some patients alternation is first observed when the fatal termination is not far distant. A woman aged forty-eight, who had suffered for years from exophthalmic goitre, eventually became markedly dropsical from cardiac dilatation, and developed bronchitis a few days before her death. In the terminal phases of her illness extrasystoles were noted, each being followed by an alternating pulse.

Treatment.—The treatment is that of the graver forms of chronic heart failure, and the reader is referred to Chapter XXI. The only further observation we would make is that alternation is not a contra-indication to the administration of digitalis or of strophanthus.

CHAPTER IX

TONICITY

THE tonicity, tone, or tonus, of the heart is a function regarding which we have but little accurate information. The classic observations of Gaskell¹ form the basis of our knowledge. Perfusion of the beating heart with alkaline solutions induces less and less complete relaxation until eventually there is systolic standstill. With acid solutions, on the other hand, the contractions become progressively enfeebled until the heart stands still in complete relaxation. These observations have been accepted as evidence that the walls of the cardiac chambers in diastole are not fully relaxed and flaccid, but have the property of tonicity whereby they offer resistance to stretching. Evidence of cardiac tonicity is also based upon the variations in the degree of cardiac relaxation under a constant venous pressure ; on the slow, periodic variations or tone-waves arising in the heart muscle apart from its systolic contractions ; and on its slow positive electric change under vagal stimulation.

The tonicity of the heart is usually measured by its diastolic volume, diminution or increase of volume without alteration of venous inflow signifying respectively increase or lowering of tonicity. Moreover, since Mackenzie² suggested that pathological dilatation of the heart was a sign of its defective tonicity, this theory has been widely accepted.

But many physiologists now doubt whether the heart possesses the property of tonicity, and others, including Starling³ and Bainbridge,⁴ deny its existence. According to the

¹ Gaskell, W. H., Schäfer's Text-book of Physiology, Edin. and Lond., 1900, ii., 203-221.

² Mackenzie, J., *Brit. Med. Journ.*, 1905, ii., 1689.

³ Starling, E. H., *Linacre Lecture on the Law of the Heart*, Lond., 1918.

⁴ Bainbridge, F. A., *Physiology of Muscular Exercise*, Lond., 1919, 48.

latter, the balance of evidence is against the existence of diastolic tone, the heart during diastole is supposed to be relaxed completely, and its filling to be determined entirely by the venous inflow. Starling uses the term "tone" in relation to the heart as signifying its ability to develop energy. In this sense "tone" is synonymous with contractility, good "tone" implying a forcible contraction, not lessened diastolic relaxation. The evidence against the existence of cardiac tonicity is, however, not altogether convincing, and when we use the terms tonicity, tone, or tonus, we do so in the ordinary acceptance of the terms, and do not refer to contractility.

When the heart is called upon for increase of work, as during muscular exercise, the cardiac output may rise from 5-8 to twenty-four litres per minute, and the volume of the heart, at first, becomes increased, because the contractions of the skeletal muscles and of the diaphragm, raising the intra-abdominal pressure, drive more blood through the veins into the heart, while the peripheral resistance is increased. It is the volume of the ventricles at the beginning of contraction rather than the pressure within them which determines the strength of contraction. Dilatation of the cardiac chambers implies stretching of their muscle fibres, and this, occurring in response to physical exertion, is beneficial when within physiological limits, because the heart in common with all other muscle has the property of developing energy in direct proportion to the length of its fibres. In health, physical exertion causes merely a temporary and physiological dilatation of the heart. The rise of arterial pressure induces a more abundant flow of blood through the coronary arteries; the cardiac muscle fibres receive a more ample supply of oxygen and of food material; and the heart, although doing more work, regains its normal volume. Thus, if the tonicity of the heart is good its chambers do not become dilated by physical exertion. But if the tonicity is lessened by fatigue or by disease, the dilatation of the cardiac chambers and the lengthening of their muscle fibres induced by physical exertion may persist, this being an adaptation to maintain an efficient circulation. If tonicity falls still lower, physical exertion causes the muscle fibres of the heart to become stretched beyond their optimum length; they have then to contract at a mechanical disad-

vantage, the systolic output lessens, the residual blood increases, the volume of the heart is persistently increased and the clinical condition is that of dilatation of the heart.¹

Depression of tonicity is probably never solely the result of physical exertion. In other words, a healthy heart does not suffer dilatation by physical stress. Unwonted physical exertion or severe exertion, long continued and oft repeated, may however be the apparent cause of dilatation if the tonicity of the heart be lessened. The condition which then results, and which is sometimes known as cardiac over-strain, is very rare in young individuals, but may be observed in middle-aged or elderly persons who have made some severe effort when they were untrained or debilitated, for example, by a recent attack of influenza. In others, without any history of violent or long-continued physical strain, cardiac dilatation often develops as a direct consequence of tonicity being lowered by toxæmia, with or without fever, as in the course of enteric fever, pneumonia and other acute infective diseases, in anæmia, in chronic alcoholism and in myocarditis. Toxæmia more often depresses tonicity than conductivity or any other function of the heart muscle. Tonicity is probably depressed by vagal stimulation, though this is disputed by some writers; the subject is one of some importance because the beneficial action of digitalis in heart disease is mainly effected by stimulation of the vagus.

The Symptoms of Depression of Tonicity vary according to the degree of depression. If this is pronounced, the reserve power of the patient's heart is so much lowered, and he usually becomes so breathless, that he is confined to bed. The heart becomes dilated, the apex impulse is displaced outwards and is diffuse, the area of cardiac dulness is increased transversely, and evidences of general venous engorgement and dropsy make their appearance. The mitral or the tricuspid valve, or both, may become incompetent; the pulse is unduly rapid, but rhythmic, usually of small volume, and alternating if the contractility of the heart muscle is also depressed. In grave cases the heart sounds have a canter rhythm.

In lesser degrees of depression, we find evidence mainly of

¹ Wiggers, C. J., *Arch. Intern. Med.*, 1921, xxvii., 475.

lack of tone of the muscle sphincters around the tricuspid or mitral orifices. For example, during the course of any acute febrile disease such as influenza, pneumonia, or rheumatic fever, a soft systolic murmur, which often amounts only to an impurity of the first sound, may develop. The murmur disappears during convalescence when the sphincter, regaining its tonicity, again closes the orifice during systole. The transient tricuspid or mitral systolic murmur, audible for a short time after slight physical exertion in anæmic or otherwise debilitated individuals, is similarly due to defective tonicity of the sphincters around the auriculo-ventricular orifices. We find this condition very often in patients, in whom the pulse rate is somewhat accelerated, during their convalescence from dysentery, malaria, or diphtheria. When the patient regains his general health the tonicity of the tricuspid and mitral sphincters becomes restored, and the first sound is again closed, even after exertion.

In rare instances the pulmonary valve may become incompetent by the muscle fibres of the conus arteriosus losing tonicity, as was demonstrated in 1880 by G. A. Gibson.¹ The diastolic "murmur of high pressure in the pulmonary artery" occasionally associated with mitral stenosis is sometimes known as the Graham Steell murmur, for it was he who drew special attention to it in 1889.² Still more rarely aortic incompetence may arise from defective tonicity of the sub-aortic musculature, although the aortic valve is healthy.

Undue pulsation of the cervical veins may sometimes be due, as Mackenzie³ suggested seventeen years ago, to defective tonicity of the bands of muscle in the wall of the right auricle which normally close the orifice of the superior vena cava. The great distension of the cervical veins which can be produced in some patients by applying firm pressure with the hand over the liver may also, we suggest, be due in part to defective tonicity of the dextral chambers of the heart. By compressing the engorged liver, we empty it as we would a sponge; the blood is driven into the vena cava and thence

¹ Gibson, G. A., *Edin. Med. Journ.*, 1880, xxv., 982.

² Graham Steell, *Text Book on Diseases of the Heart*, Manchester, 1906, 105.

³ Mackenzie, J., *Brit. Med. Journ.*, 1905, ii., 1689.

into the chambers on the right side of the heart. If their tonicity be lowered they become dilated by the greater volume of venous blood, and the jugular veins stand out, engorged, until the pressure upon the liver is withdrawn.

The Treatment of patients in whom the tonicity of the heart is defective is discussed in Chapter XXI. The defect may be transient and recoverable, as in anæmia or in the acute infections, or may be permanent as in chronic myocarditis and in the late stages of valvular disease. The defect always necessitates avoidance of any undue physical exertion. In the more severe grades of depression, the patients should be confined strictly to bed. In the lesser grades of depression the patient may usually be permitted to take any form of gentle exercise provided it does not render him breathless. Each case must be judged on its merits. Schoolboys and young adults should not indulge in football, rowing, or any other of the more strenuous forms of sport until the heart has fully regained its tonicity. The administration of iron and of strychnine is often helpful. It has long been held that small therapeutic doses of digitalis have a "tonic" action upon the heart, but the beneficial effect in cases of cardiac dilatation need not necessarily be ascribed to digitalis directly increasing the tonicity of the heart muscle. By lowering the heart's rate, digitalis gives it longer periods of diastole in which to rest and thereby all the properties of the muscle, including tonicity, become enhanced. The raising of the heart's tonicity may thus be an indirect, not a direct, effect of the drug.

CHAPTER X

AURICULAR FLUTTER

AURICULAR flutter is the condition in which the rhythmic, co-ordinate contractions of the auricular muscle are greatly accelerated, at a rate usually between 250 and 300 per minute. In our own cases the highest rate was 377, but the limit may be even higher. The auricles are contracting with extreme frequency and great regularity. The ventricles may respond to each auricular stimulus, whereby the rate of the whole heart is about 280 per minute, or the ventricles may respond only to every second, third, or fourth auricular stimulus (Figs. 142, 143, 144), giving a pulse rate of about 140, 93, or 70 respectively. Flutter may be transient, paroxysmal, and recurrent, or it may continue for months or years. The onset is always abrupt. It may cease abruptly, the heart resuming its sinus rhythm, or the flutter may be replaced by fibrillation.

Flutter was first demonstrated by one of us¹ in 1905 in a case of complete auriculo-ventricular block, the rate of the rhythmic auricular contractions being about 275, whereas that of the ventricles was about 30. In the following year G. A. Gibson² recorded a second case, with an auricular rate of 200 to 350, and a ventricular rate of about 40. Two years later, Morison³ recorded a third case. Jolly and one of us⁴ were the first to obtain electrocardiographic records of the disorder in 1909, and recognizing its essential similarity to the flutter described by MacWilliam⁵ in 1887 as a result of

¹ Ritchie, W. T., *Proc. Roy. Soc. of Edin.*, 1905, xxv., 1085.

² Gibson, G. A., *Brit. Med. Journ.*, 1906, ii., 1113.

³ Morison, A., *Lancet*, 1909, i., 39, 77.

⁴ Jolly, W. A., and Ritchie, W. T., *Heart*, 1910-11, ii., 177.

⁵ MacWilliam, J. A., *Journ. of Physiol.*, 1887, viii., 296; *ibid.*, 1888, ix., 345.

weak faradization of the mammalian auricles we termed it auricular flutter. The condition was fully considered by one of us¹ in 1914, when fifty-five cases were analyzed and discussed. Since then other cases have been recorded in this country, in France and in America. The condition has also been designated auricular tachysystole or auricular tachycardia, but the term flutter is generally adopted.

Etiology.—The disorder is comparatively rare. Only one case was observed in a series of nearly 2,500 soldiers admitted to hospital during a period of two years for affections of the heart, and in over 21,000 soldiers admitted to the medical division of an hospital in the course of a year. We have observed thirty-three cases, Mackenzie² has examined thirty, White and Stevens³ mention twelve cases. Although the number of cases that have been recorded is still small, flutter is certainly more common than would appear from statistics. In many instances the true nature of the disorder is not recognized, as many patients are so seriously ill that a detailed examination and precise diagnosis cannot be made. The age incidence is shown in the following table:—

TABLE SHOWING THE AGE INCIDENCE OF FLUTTER.

Age	1-9	10-19	20-29	30-39	40-49	50-59	60-69	70 and over	Total
Number of cases	4	8	5	11	11	19	25	3	86

The disorder is thus most frequent in later life. The youngest recorded case was aged five; the oldest, eighty-one. Seventy-eight per cent. of the patients were males.

Flutter may occur under various conditions. It is observed in the course of acute and of chronic valvular disease; in hearts that are enlarged and obviously diseased, and in hearts normal in size and apparently healthy. In more than half of the cases the arteries are thickened, and there is probably

¹ Ritchie, W. T., *Auricular Flutter*, Edin. and Lond., 1914.

² Mackenzie, J., *Diseases of the Heart*, Lond., 1913, 3rd Edit., 238.

³ White, P. D., and Stevens, H. W., *Arch. of Intern. Med.*, 1916, xviii., 712.

myocarditis as a result of disease of the coronary arteries. In about 12 per cent. there is a history of rheumatic fever, in 30 per cent. there are signs of mitral disease; in 10 per cent. there is evidence of antecedent syphilis, and in 5 per cent. there is aortic incompetence. Flutter may also arise in acute endocarditis, in the late stages of exophthalmic goitre, and in the course of diphtheria, influenza, follicular tonsillitis, and other acute infective diseases. It has also been observed during chloroform anæsthesia. In seven thousand cases of malaria, and in five hundred cases of diphtheria, we did not observe a single case of flutter.

There is no evidence that it is due to paralysis of the vagus, or to sympathetic over-action.

Morbid Anatomy.—Only a few hearts have been examined. All have been greatly dilated. Two presented chronic disease of the mitral and aortic valves; in a third, the aortic valve and coronary arteries were thickened and calcareous; another presented mitral and tricuspid stenosis and chronic interstitial nephritis. In two instances the pericardial sac was obliterated by chronic inflammation; acute pericarditis was found in another case.

In all the hearts hitherto examined the auricular walls were involved in an acute or subacute myocarditis as a result, usually, of endocarditis or of disease of the coronary arteries. In three of our cases the auricular musculature was fibrosed and infiltrated with lymphocytes and other inflammatory cells. The auricular muscle fibres may lose their striation and the nuclei remain unstained, or the fibres may undergo fatty degeneration. The sinus node may be involved in the inflammation, but it may be healthy while other portions of the auricular walls are affected. The auriculo-ventricular node and bundle, and the branches of the latter, are usually perfectly healthy. In elderly persons, evidence of chronic myocarditis is usually found in the ventricles. The lungs and other viscera are in a state of chronic venous congestion.

Experimentally, flutter may be induced in the mammalian heart by weak faradization of the auricular wall. The rapid series of weak contraction waves, originating in the stimulated area and passing over the auricular walls, was first described

by MacWilliam¹ in 1887. Both experimental and histological research indicate that in flutter an excessively rapid rhythm is developed in the auricles, in consequence of an irritative lesion in the septum, in the auricular walls, or in the appendices. In experiments, flutter may persist after the stimulation has been discontinued. The observations of Mines² on circus movement offer a possible explanation of this phenomenon. In circus movement, the contraction wave induced by a single stimulus passes repeatedly around a ring of auricular muscle, because the refractory period of the muscle has become shortened. Lewis,³ adopting this hypothesis, states that when the auricles flutter a single wave circulates continuously because the refractory period is reduced and the rate at which the excitation wave travels is usually lessened.

From the researches of Rothberger and Winterberg,⁴ Korteweg,⁵ and Canby Robinson,⁶ we know that when an increasing strength of stimulation is applied to the dog's auricles, coarse fibrillation may be superadded to the flutter, or the latter may cease while fibrillation, which ultimately becomes finer, persists. Transitions between flutter and fine fibrillation can be brought on experimentally by vagal stimulation, and the same sequence of events is often observed clinically when digitalis is given to patients with fluttering auricles. Conversely, auricular fibrillation may be replaced by flutter when quinidine is administered (*see* p. 197).

Clinical Features.—Many of the patients have, for some years, been subject to the occasional and momentary sensations accompanying an extrasystole or a short series of extrasystoles (*see* p. 93), but otherwise have been in fair health.

A paroxysm of auricular flutter begins suddenly, without

¹ MacWilliam, J. A., *Journ. of Physiol.*, 1887, vii., 296; *ibid.*, 1889, ix., 345.

² Mines, G. R., *Journ. of Physiol.*, 1913, xli., 349.

³ Lewis, T., *The Mechanism and Graphic Registration of the Heart Beat*, Lond., 1920, 341; *Lancet*, 1921, i., 551, 590.

⁴ Rothberger, J., and Winterberg, H., *Arch. f. d. ges. Physiol.*, 1910, cxxxi., 387.

⁵ Korteweg, A. J., *Proefschrift*, Leiden, 1913.

⁶ Robinson, G. Canby, *Journ. of Exper. Med.*, 1913, xviii., 704.

warning and often without any obvious cause, and lasts for a period varying from a few minutes to many months. One of our patients, a lad of seventeen, dated his attacks from a fright which he had received at the age of twelve on being chased by a drunken man. In another patient, aged twenty-one, the attacks were apparently brought on by running upstairs or by a wrestling bout. A third patient ascribed his flutter to influenza two months previously. The flutter may stop abruptly and the normal sinus rhythm be restored. The flutter may subsequently recur in paroxysms from time to time. In other patients flutter is the precursor of fibrillation. When flutter arises in a heart which is apparently healthy it usually causes distressing symptoms. More often it arises in patients whose hearts are known to be diseased, and in whom a valvular lesion or arterio-sclerosis with some degree of heart failure has been recognized. In these patients the onset of flutter ushers in urgent symptoms of heart failure.

An extreme auricular acceleration, even of 280 or 300 per minute, does not, of itself, cause any obvious symptoms. These result only if the ventricular rate becomes markedly excessive. The ventricular rate, however, does not usually exceed 140 or 150, the auriculo-ventricular ratio being 2 : 1. When the ventricles contract at this rate their period of systole is shortened, lasting for about 0·24 second, as compared with the normal 0·30 second. But diastole is still more curtailed, for it only lasts about 0·14–0·17 second, one-third of the normal. When the ventricles are stimulated to contract after so brief a diastole their contractility is not yet fully restored, and their contractions are necessarily enfeebled.

Auricular Flutter in an apparently healthy Heart.—The severity of the symptoms depends on the ventricular rate and the duration of the attack. If the ventricles respond to each auricular contraction and their rate approaches 300 per minute, the patient usually faints at the very outset. One of our patients, while playing golf, fell on the teeing green and lay unconscious for a few moments, but afterwards finished the round. Another of our patients, coming out of a warm bath, fainted, fell, and lay unconscious and on the verge of death for ten minutes before assistance could be obtained.

One of Mackenzie's patients lay unconscious for several hours, and was thought to be dead on several occasions.

Fortunately the ventricles seldom attain so critical a rate of contraction, but only respond to each alternate auricular stimulus; thus the ventricular rate does not exceed 150 (Fig. 133, *a*). The reason for this is not fully understood, but it is probably due, as suggested by Heard and Strauss,¹ to prolongation of the refractory period of the ventricles. The earliest symptom is usually sudden, severe palpitation. This is afterwards accompanied by symptoms of heart failure—præcordial discomfort or pain, giddiness, dyspnœa, anxiety and distress, and often insomnia, weakness and prostration, especially if the flutter persists and the ventricular rate remains about 140 or 150. A few patients, probably those with an excellent reserve power, suffer remarkably little except from palpitation, and continue their ordinary pursuits for several days or weeks; but even in these patients the symptoms become more urgent the longer the flutter persists.

The patients look ill. The pulse is weak and so very rapid (140 to 160 per minute) that there is difficulty in counting it (Fig. 133, *a*). It is usually perfectly regular, the auriculo-ventricular ratio being 2 : 1. In some patients the pulse is irregular, the ratio of auricular to ventricular systole varying between 2 : 1, 3 : 1 and 5 : 1 (Fig. 147); indeed the pulse may be so irregular as to simulate auricular fibrillation. Examination of the heart reveals no special features except the greatly accelerated rate of the ventricles, the rhythm of the cardiac sounds resembling those of the foetal heart. If the attack be prolonged, a soft systolic murmur may develop at the tricuspid and mitral orifices owing to dilatation of the heart; the right heart and the liver may become considerably enlarged. The jugular veins do not become grossly distended and pulsatile as they do in paroxysmal nodal tachycardia.

The attack may last for a few minutes, or for some hours, days, or weeks, and then end suddenly and spontaneously. When the flutter ceases and the normal rhythm of the heart is restored, the ventricular rate falls abruptly to 75 or 80, the

¹ Heard, J. D., and Strauss, A. E., *Arch. of Intern. Med.*, 1917, xx., 409.

patient is quickly relieved of palpitation, dyspnœa, and all other distressing symptoms, the heart and liver regain their normal size, and within a day or two the patient is able to be out of bed and may be able to undertake a fair amount of exercise without feeling the least harm. His recovery is apparently complete. The attacks tend, however, to recur



FIG. 133.—AURICULAR FLUTTER.

Arterial pulse tracings. In *a* the ventricles are responding to every second auricular beat, and the pulse is frequent and alternating. In *b* the ventricles are responding to every fourth auricular beat.

at intervals varying from days to months or years. The first attack is usually the shortest.

Auricular Flutter in the Course of Chronic Heart Disease.—

It is in patients who are known to have chronic myocardial or valvular disease that flutter is most often observed, and is most serious. The patient has usually been able to follow his ordinary avocation, with some limitation of his reserve

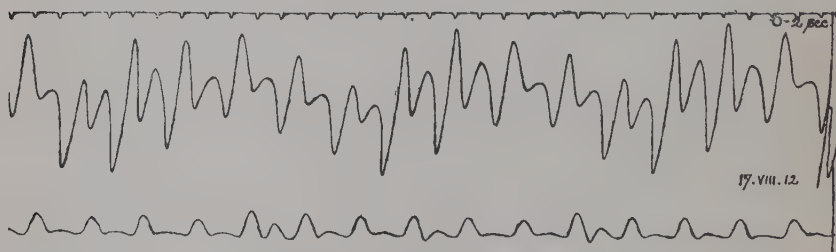


FIG. 134.—AURICULAR FLUTTER.

The ventricles respond to every second auricular beat. The pulse rate is 136.3 per minute. Jugulo-carotid and brachial tracings.

power, until the flutter starts. It may come on without apparent cause, or in consequence of strain, fatigue, indigestion, emotion, worry, a drinking bout, or some acute infection such as bronchitis or influenza. The patient soon becomes acutely ill with palpitation, dyspnœa, and other urgent symptoms of cardiac failure as described above. In a day or two

he becomes markedly cyanosed, the liver enlarges, and deep pressure over the organ causes tenderness, he has to lie propped

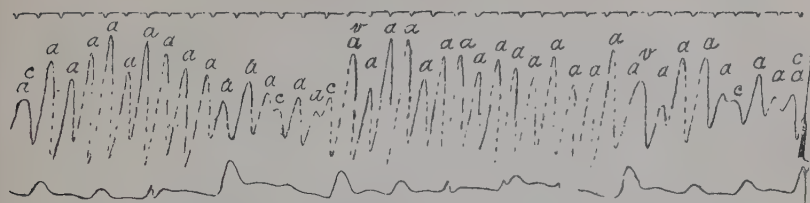


FIG. 135.—AURICULAR FLUTTER AT A RATE OF 350 PER MINUTE.

The ventricular responses to every third auricular beat are of unequal strength. Jugulo-carotid and brachial tracings.

up in bed, the bases of the lungs become œdematous, the urine is scanty and albuminous, the skin often acquires an

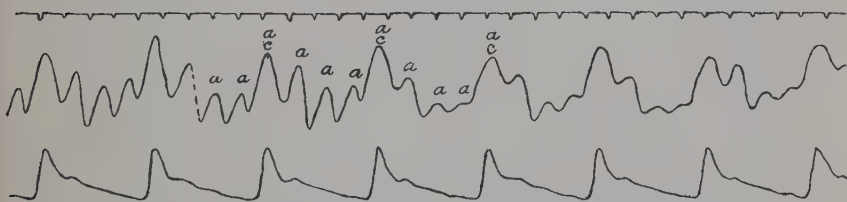


FIG. 136.—AURICULAR FLUTTER AT A RATE OF 266 PER MINUTE.

The ventricles respond to every fourth auricular beat. Jugulo-carotid and brachial tracings.

icteric tint, and the sputum may be deeply blood-stained from pulmonary congestion or infarcts. In the course

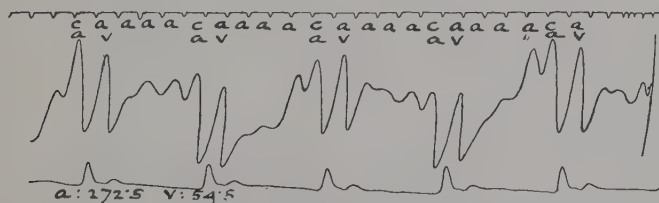


FIG. 137.—AURICULAR FLUTTER.

The ventricles respond to every fifth auricular beat. Jugulo-carotid and brachial tracings.

of a few days the patient may become somnolent, restless, or even mildly delirious, and Cheyne-Stokes breathing may occur. The condition may seem to be hopeless. Never-

theless an almost complete recovery may ensue if the flutter ceases.

Even though the mitral or aortic valves are grossly diseased, systolic murmurs become much obscured, and diastolic murmurs disappear, when the ventricular rate is 150 and when each ventricular diastole is curtailed to about 0.14 second. A presystolic murmur will likewise disappear, not merely because of the shortened ventricular diastole, but also because the flutter precludes any definite rise of intra-auricular pressure immediately before ventricular systole.

The application of pressure upon the vagus nerve about the level of the thyroid cartilage, although not retarding the auricles, usually retards the ventricles very markedly for a short period. This effect is due to the temporary depression of conductivity in the bundle, whereby only every eighth or tenth auricular stimulus passes to the ventricles (Fig. 145). The bundle escapes from the vagal inhibition soon after the pressure is withdrawn, and the rapid ventricular rate is resumed. The right vagus is usually more effective than the left in slowing the ventricles.

The arteries are usually thickened, and the blood pressure is raised. The pulse is small, rhythmic and rapid, with a rate usually of 140 to 150, the ratio of auricular to ventricular systole being 2 : 1. The pulse is often alternating as in Fig. 133, *a*, and dicrotic. Sometimes the pulse rate is suddenly doubled, or halved. It may, for example, change from 80 to 160 when the patient sits up in bed. This feature is almost characteristic of flutter. From time to time the ratio of auricular to ventricular systole may be 1 : 1, and on these occasions the pulse rate is 250, 300, or even higher. If the auriculo-ventricular ratio is inconstant, the pulse is not so rapid, but it is markedly irregular. A tracing, however, will show that the pulse-beats occur in groups of uniform duration, the length of the groups bearing a definite relation to one another and corresponding to multiples of the constant inter-auricular period (Fig. 138).

The pulsations of the jugular veins do not appear excessive, but they are masked by the respiratory movements and especially by the contractions of the sterno-mastoid muscles during inspiration. In tracings, the jugular pulsations are

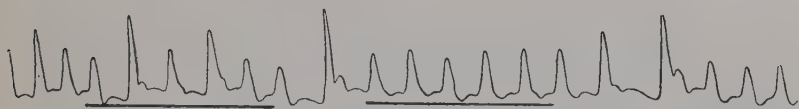


FIG. 138.—AURICULAR FLUTTER.

Arterial pulse tracing. The underlined groups of beats are of equal duration.

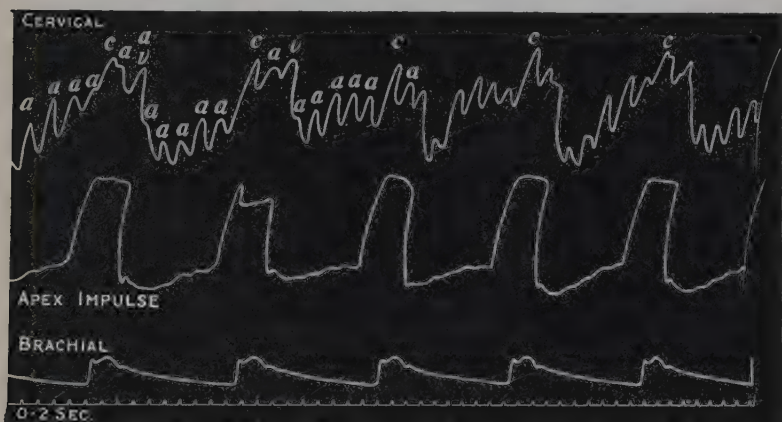


FIG. 139.—SIMULTANEOUS TRACINGS FROM A CASE OF FLUTTER AND COMPLETE AURICULO-VENTRICULAR BLOCK.

The auricular rate is 234, the ventricular rate is 32 per minute.

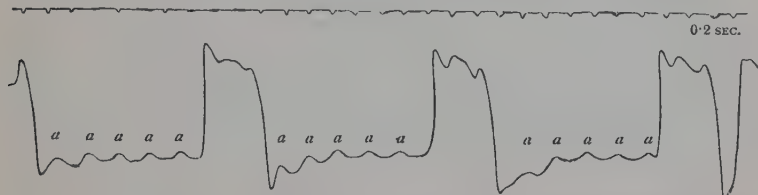


FIG. 140.—AURICULAR FLUTTER AND COMPLETE AURICULO-VENTRICULAR BLOCK. TRACING FROM APEX IMPULSE.

Auricular rate 226; ventricular rate 31.5 per minute.

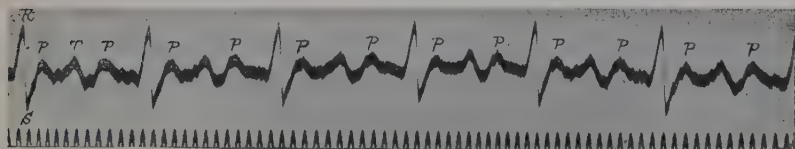


FIG. 141.—AURICULAR FLUTTER.

The ventricular rate is one-half that of the auricles. Derivation II.

of the ventricular form, the curve rising with each ventricular systole and falling when the tricuspid valve opens (Fig. 134). If an electrocardiogram can be obtained it is characteristic, especially by Derivation *III*. For each ventricular complex, which is of normal form, there are two auricular deflexions

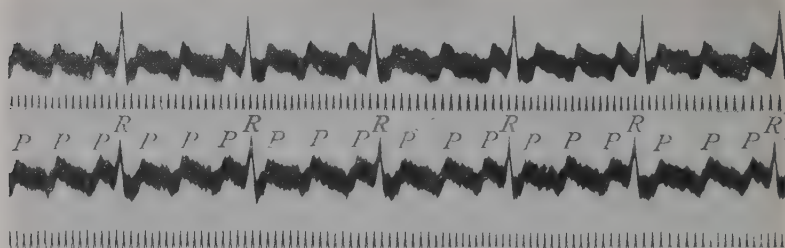


FIG. 142.—AURICULAR FLUTTER AT A RATE OF 291 PER MINUTE, WITH AN AURICULO-VENTRICULAR RATIO OF 3 : 1.

From a woman aged thirty-nine, suffering from mitral and tricuspid stenosis
Derivations *I* and *III*.

(Fig. 141). One or both may be obscured by the ventricular complex, but the auricular deflexions are clearly revealed when the ventricular rate becomes retarded by vagal compression (Fig. 145) or by digitalis (Fig. 143). The auricular

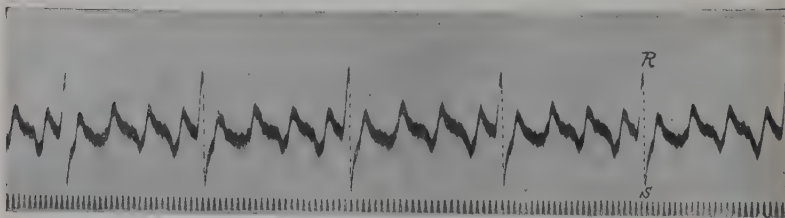


FIG. 143.—AURICULAR FLUTTER AT A RATE OF 281 PER MINUTE, WITH AN AURICULO-VENTRICULAR RATIO OF 4 : 1. DERIVATION *II*.

From a man aged forty-five suffering from heart failure.

deflexions, as recorded by a particular derivation, are all of identical form, and they are often diphasic. The initial deflexion may be either upward or downward. The auricular rate is usually fairly constant from day to day, but may vary somewhat, for example between 280 and 320.

Auricular flutter usually persists for an indefinite period,

but it may alternate, from hour to hour or from day to day, with a sinus rhythm or with auricular fibrillation. The flutter may eventually cease.

If the sinus rhythm is re-established, the patient quickly

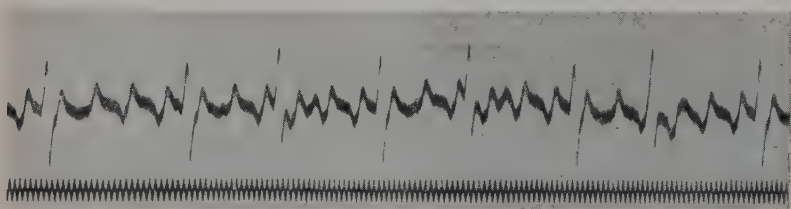


FIG. 144.—AURICULAR FLUTTER AT A RATE OF 276.4 PER MINUTE, WITH IRREGULAR VENTRICULAR RESPONSES. DERIVATION II.

and steadily improves. One of our patients was conscious of the change when the flutter ceased and the pulse rate fell from 150 to 80. All the symptoms abate; a patient who a few days previously appeared to be at death's door now lies

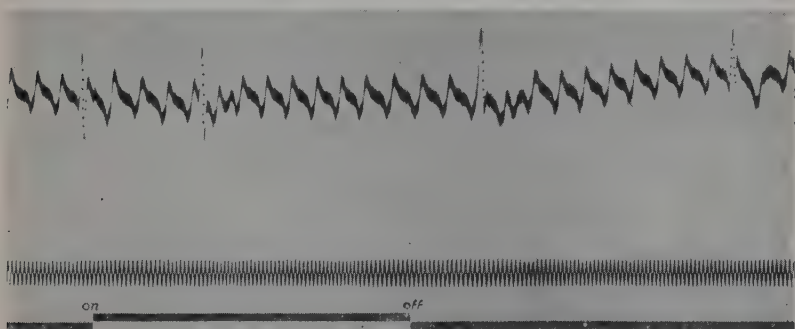


FIG. 145.—AURICULAR FLUTTER AT A RATE OF 274.2 PER MINUTE.

Compression of the right vagus during the period shown by the signal retards the ventricles but not the auricles. Derivation II.

comfortably in bed, his colour good, his breathing easy. The area of cardiac dulness lessens, the hepatic engorgement and pain pass away, the urinary flow increases, the dropsy lessens. If the flutter is replaced by fibrillation and the ventricular rate remains high, the patient's condition does not usually improve to any extent, but some are decidedly more com-

fortable and have fewer symptoms when once auricular fibrillation is established.

Auricular flutter sometimes supervenes, as a terminal event, in patients who have been confined to bed for weeks or months with grave heart failure.

Auricular flutter is occasionally observed in a heart with a partial defect of conductivity in the bundle, and the ventricles then contract in response only to every third, fourth, fifth or sixth auricular stimulus. Consequently the rate of the ventricles and of the pulse does not become much accelerated. In the case recorded by Gibson¹ the rate was about 50, in that of Hertz and Goodhart² it was 80. When there is complete

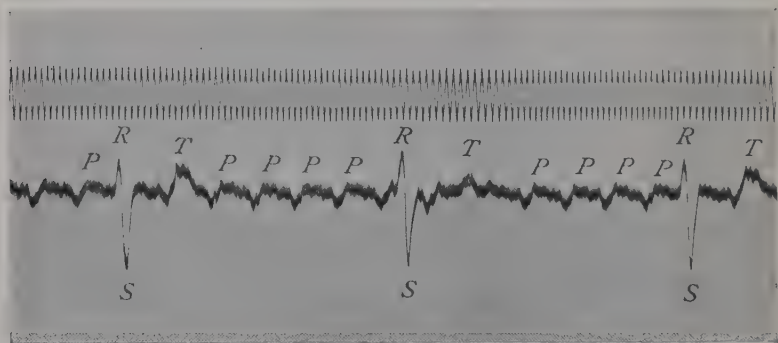


FIG. 146.—AURICULAR FLUTTER AND COMPLETE AURICULO-VENTRICULAR BLOCK.

From a man aged sixty-five. The auricular rate is 241, the ventricular rate is 37, per minute. Derivation II.

auriculo-ventricular block, the flutter does not disturb in any way the rate or rhythm of the ventricles, which continue to contract regularly at a rate of about 32 per minute³ (Figs. 139, 140 and 146). Whether the block be partial or complete, the flutter in itself causes no symptoms and will almost

¹ Gibson, G. A., *Brit. Med. Journ.*, 1906, ii., 1113.

² Hertz, A. F., and Goodhart, G. W., *Quart. Journ. of Med.*, 1908-9, ii., 213.

³ Ritchie, W. T., *Auricular Flutter*, Edin. and Lond., 1914, 88. Donzelot, *Semaine méd.*, 1914, xxxiv., 128. Vinnis, E. W. Goteling, *Ned. Maandschr. v. Geneesk.*, 1920, ix. (N.R. i.), 113.

certainly be missed unless tracings from the jugular vein, or electrocardiograms, are taken.

Diagnosis.—A paroxysmal, rhythmic acceleration of the heart, which is not due to physical exertion, and during which the pulse rate rises to 140 or more, is almost certainly due to the inception of an abnormal rhythm. Moreover, sudden and urgent heart failure, arising in the course of chronic myocarditis or valvular disease, should always arouse the suspicion of an abnormal rhythm. The use of instrumental records is not essential in diagnosis. The sudden onset, with palpitation or fainting, the continued acceleration of the pulse, at a rate of 140 or more while remaining regular, the slowing in response to vagal compression, and the characteristic sequence of events in response to digitalis are often sufficient to establish a diagnosis of flutter.

Flutter is seldom visible when the patient is examined by means of radioscopy. In only one of our cases were the rapid auricular contractions visible. In all other cases that were screened we saw merely the indrawing of the right auricular wall synchronous with ventricular systole.

Paroxysmal Fibrillation of the Auricles is excluded by the perfect regularity of the pulse. Difficulty arises when the speed of the ventricles, in response to auricular fibrillation, is so great that the irregularity of the pulse is minimal; or when, in flutter, the ventricles are irregular owing to an inconstant auriculo-ventricular ratio, as shown in Fig. 144. The latter irregularity, however, is practically never observed until the patient has taken digitalis or strophanthus for a few days. A simple tracing of the pulse may reveal the absolute irregularity characteristic of fibrillation, or the grouping of the beats characteristic of flutter (Fig. 138). If compression of the right vagus retards the pulse, the condition is almost certainly flutter, not fibrillation. A ventricular form of jugular pulse is seen in both disorders. An electrocardiogram, revealing the rapid, rhythmic, auricular deflexions, which are all essentially of the same form and comparatively large, as contrasted with the irregular and smaller deflexions of auricular fibrillation, is the final court of appeal.

In contrast to a *sinus tachycardia*, the pulse in flutter remains

remarkably constant whether the patient lies down or rises from the couch. Moreover the rate does not gradually increase when the patient undertakes some slight exertion, and is neither increased during deep inspiration nor lessened during prolonged expiration. In persons with a sinus rhythm the ventricular acceleration to 140, 160, or 180, after physical exertion such as is entailed by running a hundred yards or up a flight of stairs, gradually lessens after the exertion has ceased, and the normal rate is usually regained in one or two minutes (*see* p. 78).

No matter how ill an adult patient may be the heart very seldom maintains a rate of 140 while still retaining its sinus rhythm. Consequently a rhythmic pulse which is maintained at a rate of 140 or more in adults should always suggest the probability of flutter. This holds good not only in acute or chronic diseases of the heart, but also in patients suffering from pneumonia, exophthalmic goitre and other forms of sympathetic over-stimulation, and all acute infective diseases. We have never observed a pulse rate of 140 in typhus, malaria, or relapsing fever even when the temperature exceeded 106° F. In adults who are suffering from pneumonia a pulse rate of 140 is almost always a terminal event; we can recall only a few patients who recovered from pneumonia after their pulse rate had attained 140 per minute.

Paroxysmal Nodal Tachycardia, with a pulse rate of 140–200 or more, closely simulates flutter. But in the former the jugular veins become greatly engorged and pulsate forcibly, vagal compression does not retard the ventricles, and the electrocardiograms are distinctive.

Paroxysmal Ventricular Tachycardia, a paroxysmal series of ventricular extrasystoles, occurring at a rate of 130 to 260 per minute, seldom lasts longer than a few minutes, and the ventricles are not retarded by compression of the vagi. Polygraph tracings may aid in diagnosis, but an electrocardiogram is usually needed for precise differentiation between this disorder and auricular flutter (*see* p. 212 and Fig. 169).

Lastly, in flutter the heart's response to digitalis or strophanthus is particularly characteristic and often establishes

a perfectly clear diagnosis, even though no instrumental records are obtainable.

Prognosis.—The urgent symptoms can usually be relieved by digitalis, and when this drug has slowed the ventricular rate to 75 or 80, the patient as a rule feels wonderfully well.

It is probable that flutter always indicates myocarditis. If the patient recovers from an attack of flutter and remains well for some years the lesion in the auricular wall may be considered to have been slight, strictly limited in extent, and to be quiescent. If paroxysms recur again and again and cause grave heart failure, the myocarditis is slowly progressive and the prognosis is unfavourable. If the flutter persists and heart failure becomes extreme, the myocarditis is more widespread and probably more acute, and the prognosis is grave.

The prognosis should be based partly on the frequency and duration of the attacks, but mainly on the intensity of the dyspnœa, of the cyanosis, and of the dropsy, and on the condition of the valves, of the arteries and of the kidneys. If the heart is apparently healthy, the attack will almost certainly end in complete recovery, and the patient may not suffer from another attack for many months or years. But if the valves are diseased, if there be chronic nephritis, if the arteries are thick, or if there have been any previous signs of heart failure, the patient may recover from the paroxysm, but this will recur, and recurrent attacks are more likely to persist and to cause pronounced heart failure. The prognosis is grave if the cardiac failure persists after the ventricular rate has fallen to the normal. When flutter supervenes in a patient who already has cardiac failure he is not likely to survive for more than a few weeks; indeed he often dies within a few days.

Treatment.—*During a paroxysm* the patient should remain in bed even although the reserve power of his heart be good and palpitation be the only symptom. If he has symptoms and signs of cardiac failure, complete rest, physical and mental, is essential; and the patient needs most careful nursing, as is discussed in Chapter XXI.

Digitalis or strophanthus should be given in large doses

until the ventricular rate falls to about 75 or 80. The action of both these drugs in cases of flutter is remarkably constant and satisfactory. In an average case, without grave symptoms, twenty minims (1.3 c.c.) of the tincture of digitalis, or a corresponding dose of the drug in infusion or pill, should be given thrice daily. If the symptoms are urgent, an immediate injection of 0.5 to 1 mg. (about $\frac{1}{30}$ to $\frac{1}{60}$ gr.) of strophanthin, or $\frac{1}{500}$ gr. of crystalline digitaline, should be given intravenously, while thirty minims of digitalis tincture are administered by the mouth, this being followed by ten minims every four hours. The beneficial effects of strophanthin given intravenously are very striking. We have seen several patients, apparently at death's door, owe their recovery to strophanthin given in this manner.

In one, two, or at most three days after the patient has begun to take digitalis the ventricular rate lessens, for example, from 150 to 100. The ventricles are now responding to every third, instead of to every second, auricular stimulus (Figs. 135, 142). Sometimes the first effect noticeable is irregularity of the pulse owing to the auriculo-ventricular ratio varying between 2:1 and 3:1 (Fig. 144). Subsequently only every fourth auricular stimulus is conducted to the ventricles (Fig. 143) and the pulse rate falls to about 75 (*see* Fig. 240). Usually the patient is now relieved of all his urgent symptoms. The dose of digitalis tincture should then be reduced to about thirty minims daily, enough being given to keep the ventricular rate at about 75 or 80 and to keep the patient free from symptoms. This dose should be continued indefinitely so long as the auricles continue to flutter. If too little digitalis be given the ventricular rate will again rise and symptoms recur.

If large doses be continued the auricular flutter, which persisted even though the ventricular rate fell to 75, may be replaced by fibrillation; the ventricular rhythm will then become wholly irregular, but the rate will probably not be excessive; symptoms will not be urgent and are usually trifling. If fibrillation does ensue, all digitalis should be withheld for some days, for we know that in about half of the cases the fibrillation will cease, and the sinus rhythm will be restored temporarily. Paroxysms of flutter or fibrillation

usually recur, but in exceptional cases the sinus rhythm may be maintained for several years.

If the sinus rhythm is not regained within a week after digitalis has been stopped, its administration must be resumed, otherwise the ventricular rate will again rise too high and heart failure will recur.

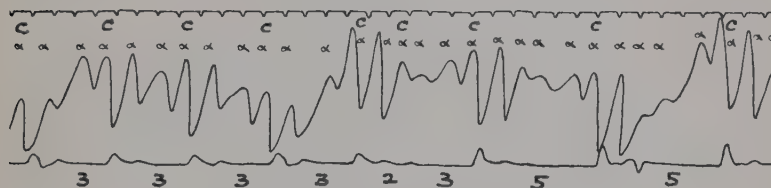


FIG. 147.—AURICULAR FLUTTER.

The auriculo-ventricular ratio is inconstant. Jugulo-carotid and brachial tracings.

In the intervals between paroxysms the treatment should be adapted to the needs of the individual patient. Even if the heart is apparently healthy, he should lead a quiet life. Hard manual labour, all physical, mental and emotional strain, and all excess of food or alcohol are injurious and may occasion further paroxysms. In other patients the further treatment is that of the causal condition, namely of endocarditis or myocarditis, whether acute or chronic, or of arterio-sclerosis.

CHAPTER XI

AURICULAR FIBRILLATION

IN many forms of cardiac disease, and particularly in chronic myocarditis and in the late stages of mitral disease, the pulse is wholly irregular in rhythm. The condition has been known for many years, and was called the "mitral pulse," "delirium cordis," "pulsus irregularis perpetuus," etc., but its true nature remained obscure until about twelve years ago. In 1906, Cushny and Edmunds¹ had pointed out the close resemblance of this irregular pulse to that which occurs in animals whose auricles are fibrillating, and they suggested that it might be due to the same cause. Mackenzie formulated the same hypothesis in 1907, but he subsequently attributed this irregularity to other causes. In 1909 Rothberger and Winterberg,² using the electrocardiograph, brought forward strong evidence in favour of the hypothesis advanced by Cushny and Edmunds, and the work of Lewis³ and of Jolly and one of us,⁴ using similar methods, proved conclusively that a wholly irregular pulse is due to auricular fibrillation.

The striking sign of auricular fibrillation is the gross irregularity of the pulse. The patient may be seriously ill with heart failure, or in fair health, the pulse rate may be fast or slow, the heart may be large or of normal size, the valves may be diseased or healthy, the arteries may be hard or soft; the one feature common to all cases is the wholly irregular pulse (Fig. 148).

¹ Cushny, A. R., and Edmunds, C. W., *Studies in Pathology*, Aberdeen, 1906, 95; *Amer. Journ. Med. Sci.*, 1907, cxxxiii., 66.

² Rothberger, J., and Winterberg, H., *Wien. klin. Wochenschr.*, 1909, xxii., 839.

³ Lewis, T., *Heart*, 1909-10, i., 306.

⁴ Jolly, W. A., and Ritchie, W. T., *Heart*, 1910-11, ii., 177.

Etiology.—Auricular fibrillation is easily produced in animals. It frequently occurs in the dying heart, and follows faradic stimulation of the auricles, ligature of a coronary artery, and perfusion of the heart with various drugs (potassium bromide, adrenalin, etc.). Vagal stimulation favours the onset of fibrillation. When fibrillation ensues the normal co-ordinate contraction of the auricular muscle ceases; the chambers dilate and the auricular muscle fibres contract independently of one another so that the surface is in a state of perpetual quivering or flickering, very similar to that which may be seen in the tongue and systemic muscles (fibrillation) in cases of progressive muscular atrophy. The contractions of the ventricular muscle remain co-ordinate, but their rhythm becomes wholly irregular, and their rate is usually accelerated. The onset of fibrillation is always abrupt, and if it ceases it does so abruptly. When once it has developed, the disorder is often permanent and continues until the heart ceases to beat; but if the stimulus is withdrawn or if the heart is perfused with a suitable fluid, the fibrillation may stop and the normal, rhythmic sequence of contractions of auricles and ventricles may reappear.

Age and Sex Incidence.—Auricular fibrillation is one of the most frequent disorders of the heart. It is seldom observed in patients under twenty years of age, and about 65 per cent. of all cases occur in patients between the ages of forty and sixty-nine. The age and sex incidence of 300 cases observed by us is shown in the following table:—

TABLE SHOWING THE AGE AND SEX INCIDENCE OF AURICULAR FIBRILLATION.

Age	10-19	20-29	30-39	40-49	50-59	60-69	70-79	80 and over	Total
Males	4	20	26	41	37	41	6	3	178
Females	2	10	21	37	23	16	11	2	122
Both Sexes ..	6	30	47	78	60	57	17	5	300

Of the 400 cases analyzed by Brachman¹ the ratio of males to females was 11 : 9, and 66 per cent. occurred after forty years.

¹ Brachman, D. S., *Lancet*, 1921, i., 374.

Co-existing Disease.—Auricular fibrillation occurs in many forms of cardiac disease, but is particularly common (1) in cases of inflammation and degeneration of the myocardium resulting from disease of the coronary arteries, which is sometimes of syphilitic origin ; and (2) in the late stages of mitral stenosis as a sequel of rheumatic infection. In a series of 70 cases of auricular fibrillation, 48 males and 22 females, we found that the incidence of rheumatism was 25 (35·7 per cent.), that of syphilis was 10 (14·7 per cent.). This agrees with the observations of Levine,¹ who considers that about one-third of the patients give a history of one or more attacks of acute rheumatism or chorea, and present signs of chronic mitral endocarditis. In mitral stenosis the maximal strain is laid on the left auricle, and disturbance of the normal auricular activity is extremely likely to ensue. Moreover in mitral disease and in degenerate, hypertrophied hearts myocardial fibrosis is often found, and may involve the auricles in particular.

Auricular fibrillation is rare in cases of aortic incompetence ; we have seen only twelve cases in the course of the last ten years. It is not uncommon in the late stages of exophthalmic goitre ; it is rarely seen in acute infective disease, but may occur as a terminal event in pneumonia and in diphtheria.

Our own histological observations and those of others² indicate that the auricular muscle is usually affected by a diffuse or patchy fibrosis, with or without cellular infiltration and other signs of subacute or chronic myocarditis. The essential lesions must be sought for in the auricular walls rather than in the primitive tissue, because the sinus node is not necessarily involved in the inflammatory process, whereas it may be inflamed although the auricular contractions are co-ordinate and rhythmic. In some cases of auricular fibrillation the auriculo-ventricular node and bundle have been found to be healthy.

Many years ago Gaskell³ suggested that fibrillar contraction

¹ Levine, S. A., *Amer. Journ. Med. Sci.*, 1917, N.S., cliv., 43.

² Schönberg, S., *Frankf. Zeitschr. f. Pathol.*, 1909, ii., 153, 462. Hedinger, E., *ibid.*, 1910, v., 296. Jarish, A., *Deutsch. Arch. f. klin. Med.*, 1914, cxv., 331. Lenoble, E., *Annales de méd.*, 1921, x., 125.

³ Gaskell, W. H., *Schäfer's Text-book of Physiol.*, Edin. and Lond., 1900, ii., 203.

of the ventricles may be brought about by a blocking in the connections between the branching muscle cells, stimuli travelling at different rates, being blocked at varying distances in the muscle and reaching the muscle cells at varying periods. In 1912 one of us advanced the hypothesis that auricular fibrillation is likewise due to defective conductivity in the auricular muscle. Vagal stimulation is known to promote and intensify auricular fibrillation, this effect being probably due to depression of conductivity, with resultant blocking of impulses in the auricular muscle. While transient auricular fibrillation may thus be a direct effect of vagal stimulation, the explanation of persistent fibrillation may be sought in the permanent defect of conductivity occasioned by chronic myocarditis. Lewis at one time considered that auricular fibrillation was the result of increased excitability of the auricular muscle, whereby ectopic stimuli constantly arose at every part of the auricular wall. This hypothesis was never generally accepted, and Lewis¹ now considers that fibrillation, like flutter, is due to circus movement (*see* p. 159), the central circulating wave, however, completing its cycle in a shorter time than that of flutter, probably because the refractory period is still more curtailed. Moreover, in fibrillation the paths of the central and of the outlying waves are rendered variable and coarsely sinuous by barriers of refractory auricular muscle.

Clinical Features.—From the clinical standpoint, cases may be divided into two groups: one in which the fibrillation occurs occasionally and in paroxysms, and another in which it is constantly present, perhaps for years. The severity of the patient's symptoms varies considerably in different cases, according to the duration of the attack, the speed of the ventricles, and the tonicity of the ventricular muscle. A paroxysm may pass off in a few minutes, or may last for hours or days and may recur at intervals, but sooner or later fibrillation becomes continuous. Fibrillation, when once established, usually persists indefinitely. It may continue for years in persons who are enjoying average health. In others, however,

¹ Lewis, T., *Brit. Med. Journ.*, 1921, i., 551, 590. Lewis, T., and others, *Heart*, 1921–22, ix., 55.

its occurrence is the signal of serious symptoms, and life may rapidly be endangered.

The onset of auricular fibrillation in experiments is always abrupt. A similar onset is sometimes observed clinically, great irregularity of the ventricles arising abruptly and causing sudden cardiac symptoms which cease when the paroxysm of fibrillation ends. The paroxysm may be of brief duration, as in the case of a man aged sixty who suddenly became very breathless when going up stairs, and found his heart to be irregular. The paroxysm of fibrillation lasted for a few hours and the auricles then resumed their normal rhythm. Another man, aged forty-seven, had two paroxysms of auricular fibril-

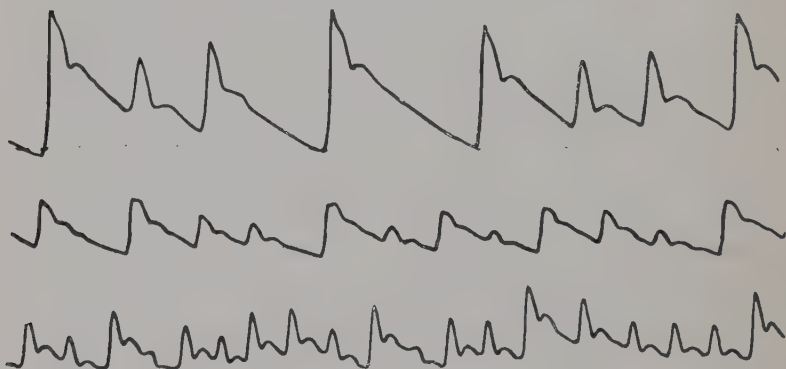


FIG. 148.—THE RADIAL PULSE IN AURICULAR FIBRILLATION.

lation while suffering from acute pneumonia. The pulse, although frequent, had been regular until the eighth day, when suddenly it presented the characteristic irregularity, the beats numbering about 120 per minute. The paroxysm lasted for eight hours, when the heart again became regular. On the ninth day the auricles passed into fibrillation for a short time, but subsequently the pulse remained regular; and convalescence, though slow, was good. Slight œdema of the feet ensued for a few days after he was allowed out of bed, but this was the only evidence of cardiac weakness throughout the illness.

If the rate of the ventricles is not excessive, and if these chambers are not dilated, the chief complaint usually is sudden

and severe palpitation and a moderate degree of breathlessness on exertion.

One of our patients suffered from rheumatic fever at the age of eighteen, and subsequently from recurrent attacks of arthritis, the mitral valve became stenosed and incompetent, and he had to abandon all forms of strenuous exertion. At the age of twenty-four, while walking quietly, he was suddenly seized with palpitation and a sensation which he described as a "cramp" of the heart. This was so severe that, although he had little dyspnoea and no dropsy, he was never again fit for his clerical work. Four months later, the heart was greatly enlarged, the auricles were persistently in fibrillation, and the pulse was wholly irregular, but its rate did not exceed 60 per minute.

In another man, aged twenty, the initial symptom was that of sudden palpitation and of a "fluttering" in the chest. He continued at work for ten days, but then became confined to bed. Four months later the sensation of "fluttering within the chest" still persisted, but he was not cyanosed, breathless or dropsical. Incompetence of the mitral valve was revealed by a systolic thrill and murmur at the cardiac apex; the pulse was wholly irregular, and the electrocardiograms were characteristic of auricular fibrillation.

In a third patient, aged thirty-four, who had contracted syphilis fourteen years previously, the first paroxysm was accompanied by sudden vomiting, breathlessness, giddiness, and a sensation of "emptiness" in the chest. He continued at work for two hours, but then had to lie down. He remained in bed for three weeks, suffering from dyspnoea and "heavy beating of the heart," but improved slowly, and a fortnight later resumed work as an engineer. In the course of the next eighteen months he had occasional paroxysms, each beginning abruptly, lasting for about a week, and ending abruptly. During these recurrent paroxysms he complained of "a fluttering like a bird inside the chest," of breathlessness and of vomiting, but never of pain. Eventually the irregularity persisted, and the pulse rate remained over 100 per minute. Three months later the feet began to swell, and in the course of the next two months he had an infarct of the lung and a thrombus in the arm.

If the ventricular rate becomes much accelerated, and particularly if the ventricles become dilated, the patient suffers from severe dyspnoea, and dropsy and other symptoms of heart failure usually follow.

A miner, aged sixty-nine, suddenly became breathless while working at the pit-head, and on the following day the feet were swollen. He was found to have mitral stenosis and auricular fibrillation; the rate of the pulse did not exceed 96, but its rhythm was grossly irregular. Relief from all symptoms was obtained by rest in bed, potassium

iodide, digitalis and theobromine sodium salicylate, the improvement coinciding with a copious diuresis. Another patient suffered from rheumatic fever when thirty-six, forty-two, and forty-eight years of age. When fifty he suffered for two days from a paroxysm of palpitation, dyspnoea and a choking sensation in the episternal fossa. Six years later, while at work, a similar paroxysm ensued, and recurred from time to time. The heart was then enlarged, its transverse dulness measuring seven inches, the arteries were thick, the pulse extremely irregular, and the veins of the neck became turgid when the hand was pressed upon the liver. A few weeks later dropsy developed and became progressive; and he died seven months after the auricular fibrillation became permanent. The left ventricle was found to be hypertrophied, the auricles were characteristically dilated, the mitral orifice measured 11 cm. in circumference; the segments of the mitral valve showed marginal thickening and the fibrosis extended into the tips of the papillary muscles. Patches of atheroma were seen in the coronary arteries, in the transverse part of the aorta and in its main branches; the viscera showed chronic venous congestion.

In the majority of cases the onset of symptoms is less abrupt, and the duration of the fibrillation cannot be ascertained precisely because the patients are unconscious of the irregularity. But even in these cases there is some limitation of the reserve power of the heart, and it is during a period of cardiac insufficiency that the auricular fibrillation is first recognized by the physician. In these cases the fibrillation is almost invariably continuous, and the intensity of symptoms depends mainly on the speed of the ventricles. If their rate is normal, or but little accelerated, the symptoms are often slight or even trivial. For example, a labourer, aged forty, presented signs of arterio-sclerosis, chronic mediastino-pericarditis and auricular fibrillation. The heart was much enlarged, and the apex impulse was fixed in the anterior axillary line; but the pulse rate was 92, he had no dropsy or other signs of heart failure, and the urine was normal. Although he came to the out-patient department of the hospital for advice, he declined to be admitted on the ground that he was not sufficiently ill.

A woman, at the age of twenty-six, was told by her doctor that her heart was affected. At the age of thirty-nine she suffered from a sharp gastro-enteritis for a few days, and became breathless; when she was admitted to hospital one month later the feet were swollen. The heart was then greatly enlarged, the apex impulse being in the sixth space, and the left border, as shown by radiosecopy, extended outwards

to the costal margin. At the apex, a systolic and a rough diastolic murmur were heard, and the pulse was wholly irregular. The Wassermann reaction was negative. At first the ventricular rate varied from 110 to 140 per minute. Four days after she began to take one drachm of digitalis tincture daily the ventricular rate did not exceed 100; three days later it fell to 70-80, and she was well enough to return home.

If the patient's symptoms are severe, the ventricular muscle is almost certainly diseased, even though the ventricular rate is normal or only slightly accelerated. For example, a charwoman, aged sixty, who yielded a strongly positive Wassermann reaction, had suffered from palpitation for seven years, from breathlessness for three years, and from swelling of the feet for four months. For the first three days after her admission to hospital the ventricular rate did not exceed 90 per minute, but she had a marked degree of heart failure which was not relieved by digipuratum. She died nine days after admission to hospital, although the ventricular rate never exceeded 100 per minute. Another patient, a labourer, aged thirty-nine, affected with mitral stenosis and incompetence, had pronounced orthopnoea and dropsy, although the ventricular rate was usually 50 to 60 and never exceeded 85. For a time he obtained great relief from digitalis, which lowered the ventricular rate to about 40, but he gradually sank and died.

The urgency of symptoms, however, is usually proportionate to the ventricular acceleration, a rate of 140, 160 or more being accompanied by well-marked evidences of failure, whereas a normal rate may be associated with but few. Moreover, the patient's symptoms usually abate, and may disappear entirely, when the ventricular rate falls to about normal. This is illustrated by the following case.

A woman, aged fifty-one, had been told fourteen years previously that her heart was affected. She had suffered from intermittent attacks of breathlessness and of dropsy, and had been confined to bed for two months before being admitted to hospital.

The heart was much enlarged; a loud mitral systolic murmur was audible; the superficial veins of the neck and arms were swollen, standing out like cords. She was very dropsical, and the ventricular rate varied from 130 to 150. Sixty minims of digitalis tincture were given daily, and much fluid was drained off the legs by means of Southey's tubes. A week later, the ventricular rate did not exceed

130. At the end of the second week there was no material change, but during the third week, when the ventricular rate fell to about 110, the dropsy almost entirely disappeared. The dose of digitalis tincture was reduced to forty-five minims daily. At the end of the fifth week, the mitral murmur and all traces of dropsy had disappeared. In the sixth week, when the ventricular rate only once exceeded 100, she was well enough to be allowed out of bed daily. In the seventh week, when the ventricular rate did not exceed 88, she was taking fifteen minims of digitalis tincture daily and was able to walk about the ward and on to the verandah. She was then relieved of all her urgent symptoms, although the auricles were still fibrillating.

Many cases of persistent fibrillation are repeatedly in hospital, breathless and dropsical. They are relieved by rest and digitalis. On their return home the drug is discontinued or taken in insufficient doses, and a few weeks later, when the ventricular rate rises to 120, 150, or more, they are again seeking admission to hospital. Thus they drift in and out of hospital for a few years.

The Pulse in auricular fibrillation is very characteristic, the beats being wholly irregular in rhythm and size (Fig. 148). The variations in the size of the pulse waves are not dependent on the duration of the preceding diastole, as in a normal heart. The rate of the pulse varies within wide limits, as already described. It is often about normal, and symptoms of cardiac failure are then usually trivial or in abeyance. The rate may be increased greatly, running about 120 or even as high as 190, and then symptoms of cardiac weakness are apparent. In exceptional cases the rate is persistently infrequent (40 to 50 per minute) owing to partial auriculo-ventricular block of organic origin. In very rare instances the ventricular rate is persistently about 32 per minute and regular, owing to complete auriculo-ventricular block (Fig. 157).

The pulse rate does not vary with respiration, and is practically never retarded by compression of the vagus.

In auricular fibrillation some degree of partial block in the bundle can often be induced, with great benefit to the patient, by the administration of digitalis or strophanthus, but the ventricular rate again becomes accelerated after the drug has been discontinued.

The association of fibrillation with partial block is illustrated by the following case.

A soldier serving on the Indian frontier contracted syphilis at the age of twenty. He was under treatment for only six weeks, and at the age of thirty-six was invalided out of the service because of valvular disease of the heart. Ten years later he was working as a porter. One day, while taking a hot bath, he collapsed and lay faint and semi-conscious for ten minutes until he was able to call for help. When seen a few days later he was cyanosed but not dropsical, the lungs presented emphysema and slight bronchitis, the pulse was very irregular, and its

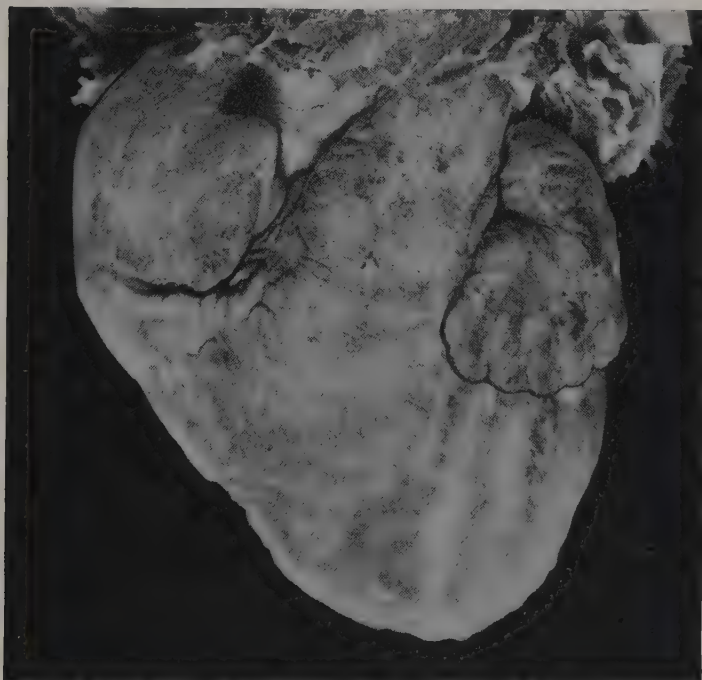


FIG. 149.—HEART OF A WOMAN AGED FORTY-THREE WHO DIED OF MITRAL DISEASE AND AURICULAR FIBRILLATION.

Dilatation of the auricles and right ventricle.

rate was 70 per minute. Under the influence of digitalis the pulse rate fell to between 45 and 50, while the irregularity persisted. This infrequent rate was at first thought to be due to the drug, but the ventricular rate remained persistently infrequent after digitalis had been discontinued, and it became evident that there was a partial auriculo-ventricular block due to organic disease of the bundle.

The rate of the pulse is often misleading because many of the ventricular beats may be missed at the wrist (Fig. 158).

It is usually in cases with a rapid ventricular rate, or in those having a coupled rhythm owing to ectopic ventricular contractions (Fig. 164), that this "pulse deficit" occurs; but in all cases of fibrillation the ventricular rate should be ascertained by auscultation.

The Heart.—When the auricles are in fibrillation they

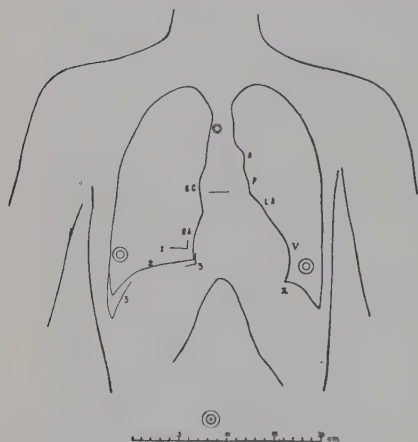


FIG. 150. — ORTHODIAGRAM. NORMAL HEART, FEMALE PATIENT, AGED THIRTY-TWO.

1. In deep expiration. 2. In tranquil inspiration. 3. In deep inspiration.
- A, Curve of descending portion of arch of aorta. P, Pulmonary curve.
- LA, Left auricular curve. V, Left ventricular curve. SC, Curve of superior vena cava. RA, Curve of right auricle. The position of the episternal notch, level of third chondro-sternal articulation, the nipples and umbilicus are shown. X indicates the position of the apex-beat.

are always dilated and the heart is enlarged (Fig. 149). The area of cardiac dulness is altered in corresponding degree, extending outwards to the right as far as, and often beyond, the right sternal border. On radio-scopic examination the dilatation of the right auricle is usually very notable, unless the patient has no symptoms of heart failure. But in nearly all cases who are under treatment for even slight degrees of failure, and especially in those who are breathless and cyanosed, the heart is seen to be much enlarged and of a more or less globular form. The right auricle bulges markedly to the right, while the

shadow of the conus arteriosus and that of the left auricle are unduly prominent to the left, and may be so large that they become fused with one another and with that of the left ventricle (Figs. 152, 153, 154). The long diameter, from the cavo-auricular junction to the apex, is on an average 16.4 cm., but may be 18 or 19 cm., as contrasted with the normal 14 cm. in men and 12.8 cm. in women. The

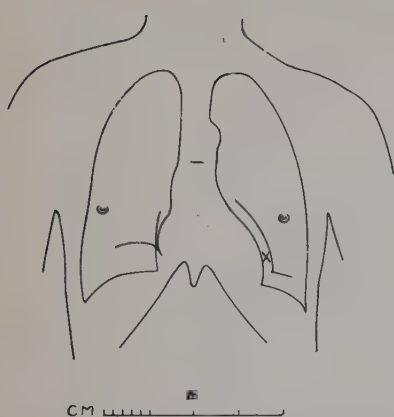


FIG. 151.—ORTHODIAGRAM. NORMAL HEART DURING FULL INSPIRATION. The borders of the right auricle and left ventricle are also shown during full expiration.

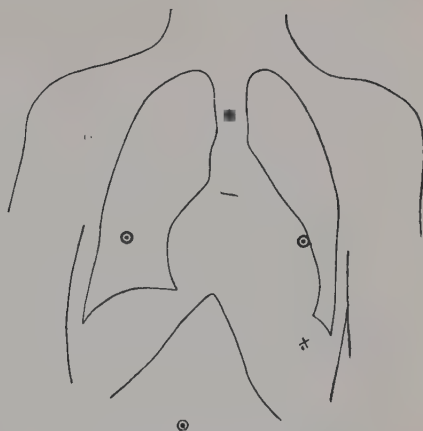


FIG. 152.—ORTHODIAGRAM. AURICULAR FIBRILLATION.

Man aged twenty-one.

average transverse measurements to right and left of the middle line are 5.8 cm. and 9.7 cm., giving a total transverse diameter of 15.4 cm., in contrast with the normal 12.5 to

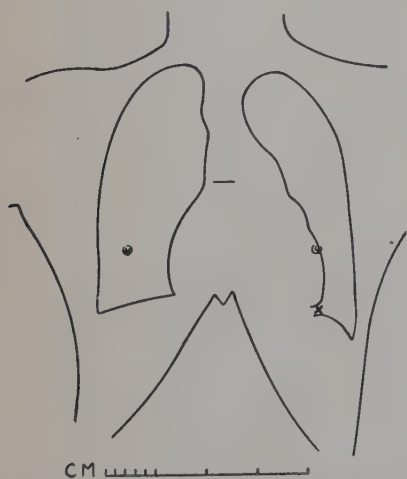


FIG. 153.—ORTHODIAGRAM. MITRAL STENOSIS AND AURICULAR FIBRILLATION.

Man aged twenty-seven.

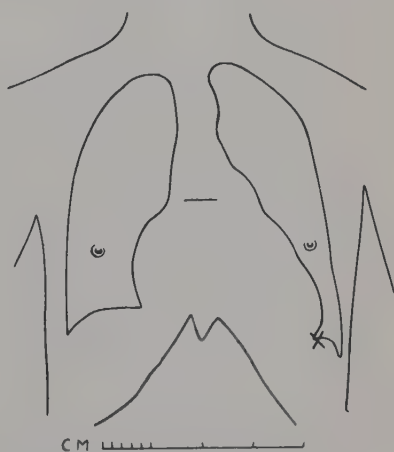


FIG. 154.—ORTHODIAGRAM. AURICULAR FIBRILLATION.

Man aged forty.

13 cm. in men and 11.5 cm. in women.¹ No auricular contractions are visible, but the margin of the right auricular shadow may be seen to be indrawn with each ventricular systole.

The Cardiac Sounds.—The complete irregularity of the ventricles is usually even more obvious on auscultation than when feeling the pulse. The sounds may be feeble or accentuated, and pure or accompanied by murmurs, according as the valves are healthy or diseased.

Of all the valvular lesions, mitral stenosis is that most often associated with auricular fibrillation. In this lesion the strain upon the left auricle is constant, as the obstruction at the mitral orifice is permanent, in contrast with the intermittent strain in mitral incompetence where the auricular congestion is rapidly relieved during ventricular diastole. The cessation of co-ordinate auricular contractions makes the characteristic, presystolic, crescendo murmur of mitral stenosis disappear, consequently the recognition of this valvular lesion is difficult unless its existence is already known. When the auricles are in fibrillation, however, mitral stenosis should be suspected if there is a mitral systolic murmur, if the second sound at the base is reduplicated, or if it is unduly emphatic at the pulmonary area. The existence of mitral stenosis may confidently be affirmed if the apical diastolic murmur is recognized.

The murmurs at the apex are extremely variable, and it is often impossible to define them when the ventricular contractions are frequent. The shortness of diastole prevents adequate filling of the ventricles, and thus minimizes systolic reflux; consequently a systolic murmur may be audible only after the longer pauses, and similarly the diastolic murmur may also be inaudible. Aortic murmurs are also variable, the systolic murmur being shorter and softer after shortened diastolic periods, and longer and harsher after longer intervals. An aortic diastolic murmur may be unrecognizable if the pulse is very frequent.

Venous Pulsations.—The degree of pulsation in the jugular veins varies in different patients. In many it is slight and inconspicuous; in others, and particularly in those with

¹ Fowler, W. Hope, and Ritchie, W. T., *Edin. Med. Journ.*, 1912 (N.S.), ix., 197.

cardiac failure, it is large, consisting of one large wave synchronous with each ventricular systole. The abnormal

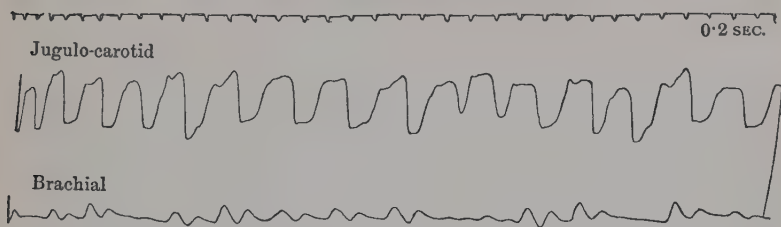


FIG. 155.—AURICULAR FIBRILLATION IN A MAN AGED TWENTY-ONE, WHO HAD BEEN BREATHLESS FOR FOUR YEARS, AFTER SUFFERING FROM RHEUMATIC FEVER.

character of the pulsation is easily studied in tracings. If the ventricular rate is frequent, the jugular tracing usually

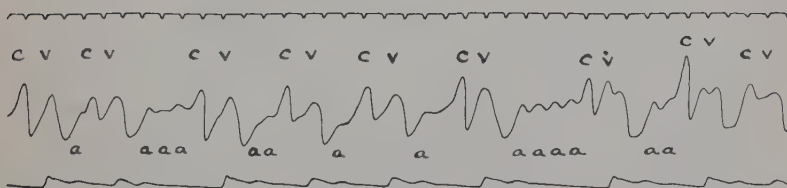


FIG. 156.—CERVICAL AND RADIAL CURVES IN AURICULAR FIBRILLATION. During diastole auricular wavelets are evident in the cervical curve.

shows a single large wave, perhaps broken up into two or more summits, commencing synchronously with the carotid

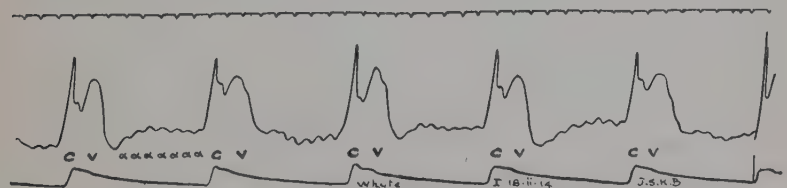


FIG. 157.—CERVICAL AND ARTERIAL CURVES FROM A CASE OF AURICULAR FIBRILLATION AND COMPLETE AURICULO-VENTRICULAR BLOCK.

During diastole auricular wavelets are seen in the cervical curve. The arterial pulse is regular at a rate of 35 per minute.

beat and ending when the tricuspid valve opens (Figs. 155, 156). The *v* wave may succeed *c* without any interval, or may be separated from it by an interval of short duration.

If the pulse rate is infrequent, a new series of small waves may be recognized during diastole (Fig. 156). These are generally small and of varying size and duration, numbering from 300 to 600 per minute, but accurate enumeration is impossible as the wavelets are lost when the ventricles are

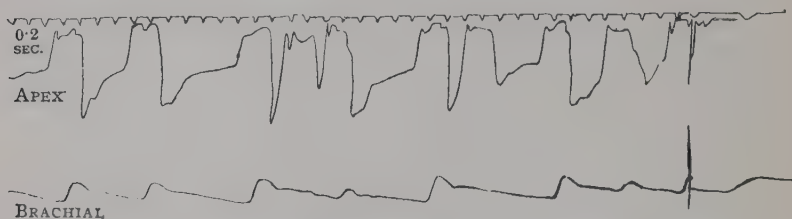


FIG. 158.—AURICULAR FIBRILLATION.

Showing a pulse deficit; the fourth and seventh ventricular beats do not cause an arterial pulse wave.

in contraction. The wavelets may be quite distinct in a tracing from the cardiac apex, but the true auricular wave is always absent in tracings from the apex-beat, from the jugular vein and from the liver.

Electrocardiograms present distinctive and characteristic features. The auricular deflexion, *P*, has disappeared and the period of ventricular diastole is occupied by a series of small deflexions, irregular in size and rhythm, comparable to the

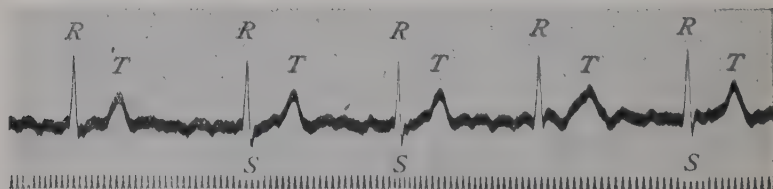


FIG. 159.—AURICULAR FIBRILLATION.

Each ventricular complex, *R*, *S*, *T*, is of normal form, but the ventricular rhythm is irregular. Small irregular deflexions are seen during ventricular diastole, namely between the end of *T* and *R*. Derivation *II*.

irregular wavelets which may be seen in the jugular tracing. These irregular deflexions, when comparatively large and not exceeding 500 or 600 per minute, represent coarse fibrillation of the auricles; when smaller and more frequent they represent fine fibrillation (Figs. 159, 160, 161).

The ventricular complexes, although varying in different patients according to the relative hypertrophy of the two ventricles, are otherwise normal (Figs. 159, 160, 161). This indicates that the ventricles are contracting in response to

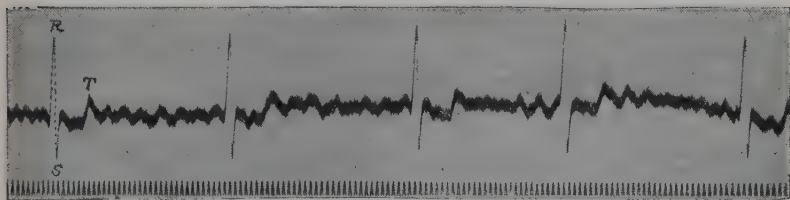


FIG. 160.—AURICULAR FIBRILLATION. DERIVATION II.

stimuli of supraventricular origin. Some of the innumerable auricular stimuli which are constantly reaching the auriculo-ventricular node are being transmitted to the ventricles, and the transmission is by the normal path. Moreover, this path

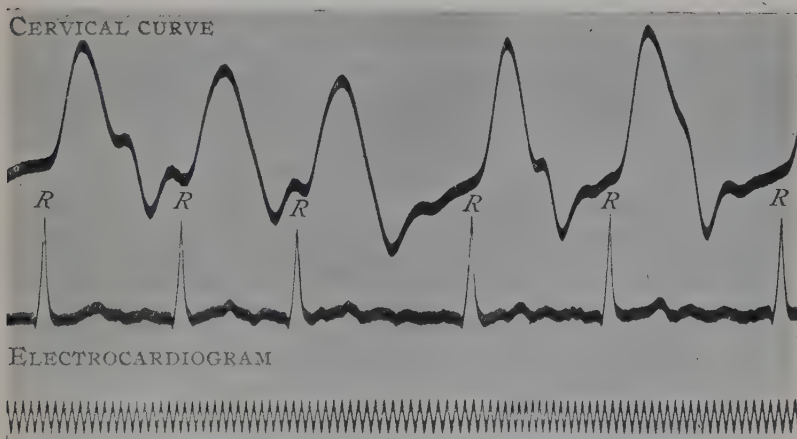


FIG. 161.—SIMULTANEOUS CERVICAL CURVE AND ELECTROCARDIOGRAM BY DERIVATION II, FROM A CASE OF AURICULAR FIBRILLATION.

Boy aged seventeen.

is intact and available, for the auriculo-ventricular node, the bundle and its branches are usually normal on histological examination.

In a few cases of auricular fibrillation the ventricles follow

their own intrinsic rhythm, beating regularly at a rate of about 32 per minute, because of complete auriculo-ventricular

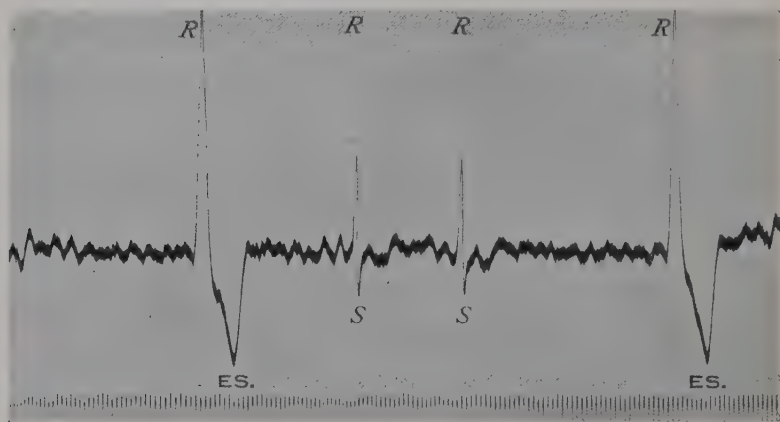


FIG. 162.—AURICULAR FIBRILLATION AND TWO VENTRICULAR EXTRASYSTOLES (ES).

Derivation II, from a man aged forty-five suffering from mitral incompetence, and very severe heart failure for two months.

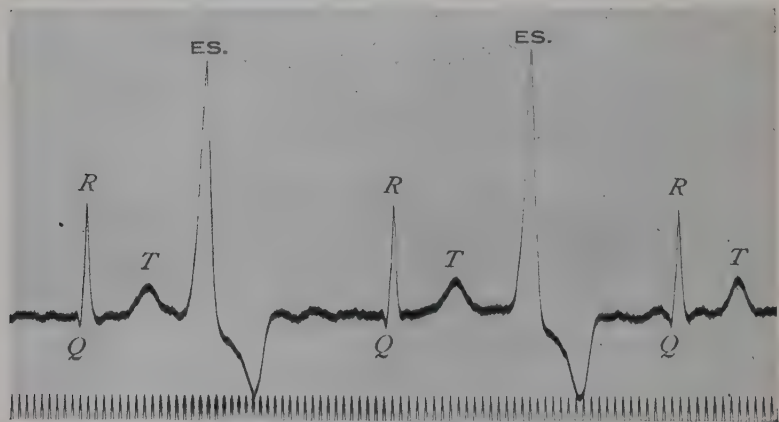


FIG. 163.—AURICULAR FIBRILLATION.

The ventricular complexes are alternately of normal form (*Q*, *R*, *T*) and ventricular extrasystoles (*ES*). Derivation III.

block (Fig. 157). This condition is comparable to the regular, infrequent, ventricular rhythm recorded by Fredericq¹ after

¹ Fredericq, L., *Arch. internat. de physiol.*, 1904-5, ii., 271.

the bundle was severed experimentally in animals whose auricles were fibrillating.

In some cases of fibrillation electrocardiograms may reveal the occurrence of superadded ventricular extrasystoles, each being represented by a large, diphasic complex identical with those described on pages 101, 102. The extrasystole may occur as an isolated event at irregular intervals (Fig. 162). In other patients it recurs with considerable regularity at a constant and short interval after each normal ventricular complex (Figs. 163, 164), and is followed by a prolonged ventricular diastole, the duration of which is wholly inconstant. The

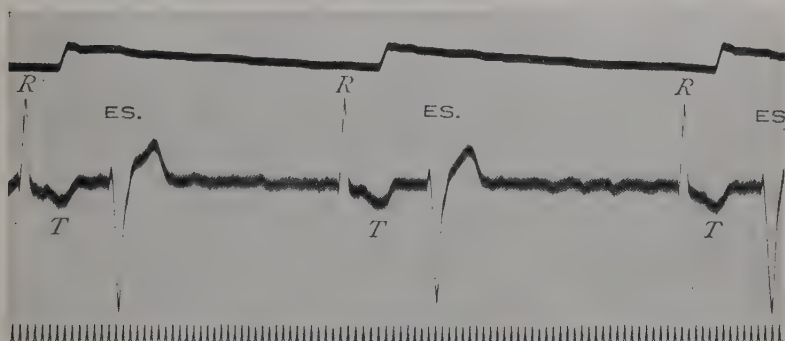


FIG. 164.—SIMULTANEOUS RECORD OF THE BRACHIAL PULSE AND ELECTROCARDIOGRAM BY DERIVATION *III*, FROM A CASE OF AURICULAR FIBRILLATION WITH REGULARLY RECURRING VENTRICULAR EXTRASYSTOLES WHICH FAIL TO SEND A PULSE WAVE INTO THE ARTERIES.

corresponding pulse beats may be coupled, or the extrasystoles may fail to send a pulse wave into the arteries, the pulse rate being therefore exactly half that of the ventricles (Fig. 164). This form of coupled rhythm is seldom observed except in patients to whom considerable doses of digitalis or strophanthus have been administered. It is a grave sign of an overdose with ventricular failure, and is an indication to discontinue the administration of those drugs at once. The occurrence of even single ventricular extrasystoles in cases of auricular fibrillation is likewise a grave sign, and few cases survive for more than two years. But one of our patients, who died suddenly at the age of sixty-four, had suffered intermittently from dropsy for ten years, and lived for more than eight years

after we first obtained electrocardiographic proof of his auricles being persistently in fibrillation and of superadded ventricular extrasystoles which sometimes gave a coupled rhythm.

Diagnosis.—There is seldom any difficulty in recognizing auricular fibrillation, for the wholly irregular pulse is characteristic. It is only when the speed of the ventricles attains or exceeds 150 or 160 that a minimal degree of irregularity is likely to be missed when feeling the pulse. The absence of any definite acceleration of the pulse during inspiration, and of retardation during expiration, and again the absence of any retardation when the right vagus is compressed, are confirmatory, but not distinctive, signs.

The wholly disorderly nature of the irregularity almost certainly excludes the possibility of it being due to abundant *extrasystoles*. But if the latter are very numerous, and especially if they are arising at different sites in the heart, some being ventricular while others are auricular or nodal, and if the post-extrasystolic pause is therefore very variable, auricular fibrillation may be simulated closely. Under these circumstances the differentiation may not be possible without instrumental aid.

Fibrillation may be simulated by *auricular flutter* with pronounced irregularity of the arterial pulse because of an inconstant ratio of auricular to ventricular contraction (Fig. 145); but the absence of any retardation of the pulse by means of pressure upon the right vagus almost certainly excludes auricular flutter.

In rare cases of *nodal rhythm*, with synchronous contraction of auricles and ventricles, the heart's action is decidedly irregular; but in these cases the jugular pulsation is usually of larger size than in auricular fibrillation. In all cases presenting difficulty of diagnosis, a polygraph tracing, or one of the arterial pulse alone, may be of great service; but the electrocardiographic evidence is the most reliable.

The prognosis in cases where auricular fibrillation has ensued is always more grave than in comparable cases with a sinus rhythm, and we have never seen a patient with fibrillation whose cardiac power was not at any rate subjectively lessened. Sir James Mackenzie states that he has

known patients lead arduous lives for as long as ten years, but such cases are certainly exceptional. One of our patients, who died at the age of thirty-six, had persistent auricular fibrillation for at least three years before his death, and enjoyed fair health for months at a time. He suffered from double mitral disease, and the first symptoms were associated with a pulmonary infarct. Another patient, whose auricles were in fibrillation, to our knowledge, for nine years before he died, worked at the pit-head for more than six of those years.

A medical colleague, who is now aged 85, has had auricular fibrillation, to our knowledge, for the last six years, and now leads a quiet life, though he remained "on duty" during the war. He has had irregularity of the heart for, he thinks, forty years.

The cases which are associated with mitral stenosis seem to have a better prognosis than those which are due to other cardiac lesions. In mitral stenosis the left auricle is overstrained, and its action is perhaps more easily disturbed. In other lesions the auricles merely share in the general stress, and fibrillation indicates a widespread disorganization of the heart muscle.

In a general way the inception of permanent auricular fibrillation is the beginning of the end, though many patients are able to lead quiet lives for a year or two after the onset.

The dilatation of the auricles, which always ensues, is of particular importance, for it seems to be a main factor in producing clotting in the appendices, and the strain of a pulmonary infarct is badly borne. Infarcts are much more common than is perhaps realized. They are generally accompanied by frank hæmoptysis or pleural friction, and by the evidences of consolidation; but we have often found more infarcts on post-mortem examination than were suspected from clinical observation, and their occurrence need not be accompanied by any obvious physical sign. A central infarct, beyond the reach of clinical examination, is a not infrequent cause of the onset of acute cardiac symptoms.

The immediate prognosis depends chiefly on the rate of ventricular contraction, the presence or absence of ventricular dilatation, and the response to the administration of digitalis. Patients differ markedly in these respects. The prognosis is

relatively favourable if the pulse rate, with or without drug-ging, can be kept within normal limits. Dropsy or other sign of dilatation of the ventricles always renders the prognosis grave ; but the patient nearly always makes a partial recovery from the first attack of dropsy. If digitalis lessens the frequency of the pulse, promotes diuresis and abolishes dropsy, the outlook is favourable for a time at least. The prognosis is always grave if ventricular extrasystoles are superadded, and these patients will probably die within two years at latest. A moderate degree of fibrosis of the bundle, causing partial auriculo-ventricular block, is beneficial in so far as it prevents the ventricular rate from becoming excessive. One of MacKenzie's patients with auricular fibrillation and complete block was working two years after the condition was recognized.

The presence of renal disease in these cases always adds greatly to the gravity of the prognosis.

Death may ensue gradually owing to progressive failure of the ventricles. It may, however, be sudden because of ventricular standstill or, as first suggested by MacWilliam in 1889, because the ventricular muscle also passes into fibrillation. In one case in which we observed this fatal event, the ventricular beats, which had been hitherto strong although irregular, ceased abruptly, the face became pallid, a brief tonic spasm of the body ensued, the pulse ceased, and the cardiac sounds became inaudible. A few gasping respiratory movements occurred after the ventricles had ceased to beat, cyanosis rapidly supervened, and a tremulous quiver, transmitted from the fibrillating ventricles to the chest wall, could be seen and felt for a few seconds in one or more of the intercostal spaces.

Treatment.—In this chapter only the essential principles are referred to, for the treatment is that of chronic heart failure, as discussed in Chapter XXI.

The danger lies in a frequent ventricular rate ; the condition of the auricles is relatively immaterial. The work performed by the ventricles must be reduced to a level at which the patient is free from dyspnœa and other symptoms. Even in mild cases the patient must desist from hard manual labour, or renounce many of his professional activities, and

lead as quiet a life as possible, rising late, avoiding all strain, whether physical or mental, which induces breathlessness, palpitation or fatigue, and retiring early to bed. In graver cases the patient should be confined to bed and be under the care of an efficient nurse. Complete recumbence is always beneficial, and the administration of digitalis or strophanthus is necessary in nearly every case.

It is in this disease that these drugs have the greatest therapeutic effect. Although they never arrest the fibrillation, they lower the conductivity of the bundle and thus lessen the number of stimuli which are transmitted to the ventricles.

ŒDEMA

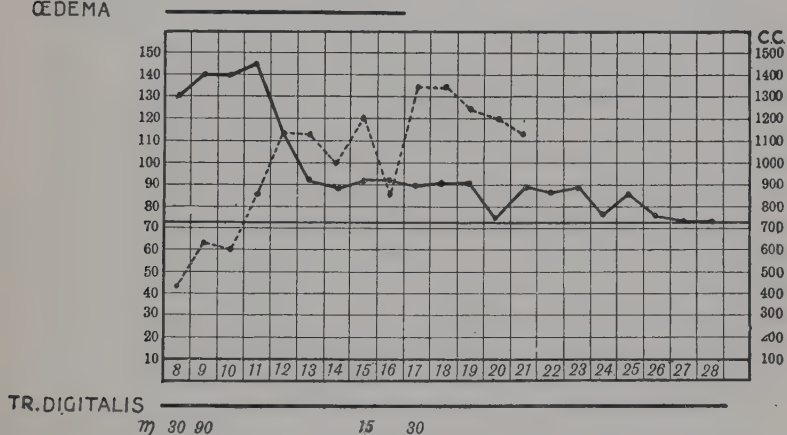


FIG. 165.—AURICULAR FIBRILLATION. RESPONSE TO DIGITALIS.

The dotted line represents the urinary output in c.c.; the continuous line, the ventricular rate. Male aged fifty-one, syphilitic myocarditis.

Consequently the rate of the ventricles and of the pulse is reduced, the ventricular contractions become more forcible, the circulation improves, and the dyspnoea and other symptoms disappear. If the patient is dropsical, the oedema disappears also, coincident with the onset of an efficient diuresis (Fig. 165).

The tincture of digitalis, and Nativelle's granules of $\frac{1}{40}$ grain of digitalin, are among the most convenient preparations. The dosage must be adapted to the needs of the individual. Many find that ten or fifteen minims of the tincture daily is sufficient to keep them free from dyspnoea and palpitation, and to

restrain the ventricular rate within normal limits. In patients who are suffering from superadded cardiac failure it is advisable to administer sixty minims (4 c.c.) of digitalis tincture daily for about one week or more, until the dropsy and breathlessness have disappeared. If the ventricular rate is 150 or more, or if urgent signs of cardiac failure arise, an initial intravenous dose of 0.5–1 mg. (about $\frac{1}{130}$ to $\frac{1}{60}$ gr.) of strophanthin, or $\frac{1}{500}$ gr. of crystalline digitaline, is often of great service. The intravenous use of strophanthin produces within an hour or two an appreciable retardation of the ventricles and amelioration of the patient's condition.

After the ventricular rate has been controlled, the daily dosage of digitalis tincture should be reduced to the minimum that is compatible with the patient's comfort and well-being. Fifteen or twenty minims of digitalis tincture is usually ample. But the drug must be administered steadily month after month and year after year; on its cessation for more than a few days the pulse becomes unduly frequent and symptoms recur. This was illustrated in a woman, at one time under our care, who had taken digitalis for seven years. Her symptoms rapidly subsided upon its administration, but they recurred on more than one occasion within a fortnight of its being stopped. The drug should be discontinued temporarily when the ventricular rate falls below 60 per minute, or if a coupled rhythm supervenes.

Quinidine Sulphate has been acclaimed as a means of restoring the normal rhythm of the auricles in cases of auricular fibrillation. If an initial dose of 0.2 gramme, given by the mouth twice daily, produces no toxic symptoms, 0.4 gramme (6 grains) may be given orally three or four times a day. Since 1918, when the use of this drug was first advocated by Frey,¹ the normal auricular rhythm is claimed to have been restored in 57 per cent. of over 200 cases. The arrest of the fibrillation is ascribed to prolongation of the refractory period of the auricles by means of the drug, which also depresses conductivity and paralyses the vagus.²

In successful cases the sequence of events produced by

¹ Frey, W., *Berl. klin. Wochenschr.*, 1918, lv., 417, 849.

² Lewis, T., and others, *Brit. Med. Journ.*, 1921, ii., 514; *Heart*, 1921–22, ix., 55.

quinidine is coarse fibrillation, impure flutter, flutter and then, after a short period of arrest of the whole heart, sudden restoration of the normal auricular rhythm.¹ During the transitional phases the ventricular rate may be considerably accelerated; extrasystolic ventricular tachycardia may occur at a rate of 150–200 per minute, as in one of our cases; and there is a risk of sudden death from ventricular fibrillation. Another danger is that when, the normal rhythm being restored, the auricles again contract portions of thrombi may be detached from the auricular walls and embolic phenomena result. Among minor unpleasant effects which may ensue upon administration of quinidine are sweating, diarrhoea and abdominal pain. Quinidine should not be administered to patients who present cyanosis, dropsy and other signs of severe heart failure. After this has been relieved by digitalis, and while that drug is still being administered, an attempt may be made by means of quinidine to restore the normal rhythm.

In only three of our twenty-three cases was the normal auricular rhythm restored. The restoration was effected within a few days by comparatively small doses; in one instance on the third day, after 2.2 grammes of the drug. In one case flutter was induced at a rate of 292 per minute. In two instances quinidine had to be discontinued, once because of extrasystolic ventricular tachycardia at a rate of 150 per minute, and in a second case because of nausea and vomiting. In the remaining cases the administration of quinidine, even in doses as large as 20 grammes within fifteen days, failed to arrest the fibrillation and, although causing no unfavourable symptoms, did not benefit the patients.

In the most favourable cases the arrest of the fibrillation is only transient. The normal rhythm is seldom maintained for longer than a few hours or days, and the general condition of the patients is but little, if at all, benefited.² In exceptional cases the normal rhythm has been maintained for several months by intermittent administration of quinidine.¹ On the whole quinidine seems to be of little therapeutic value; in efficacy it cannot be compared to digitalis.

¹ Levy, R. L., *Proc. Soc. Exper. Biol. and Med.*, 1921, xix., 88.

² Ellis, A. W. M., Clark-Kennedy, A. E., *Lancet*, 1921, ii., 894.

CHAPTER XII

NODAL RHYTHM

IN this disorder, which is also termed auriculo-ventricular rhythm, each beat of the heart consists of a synchronous contraction of the auricles and ventricles in response to a stimulus arising in the auriculo-ventricular node. Each stimulus passes backwards to the auricles and onwards, by means of the bundle and its branches, to the ventricles. The disorder may be paroxysmal and intermittent, or continuous. The rate of the heart is either (1) retarded or approximately normal, or (2) accelerated to a rate which may exceed 200 per minute. Its rhythm is usually regular. Each auricular contraction may occur simultaneously with that of the ventricles; the ventricular contraction may begin before that of the auricles; or the auricular contraction may begin before the ventricular, but with a greatly diminished interval (0.10 second) between each auricular and ventricular contraction. The disorder must not be confused with the cardiac irregularity, originally designated nodal rhythm by Mackenzie,¹ which is now known to be due to auricular fibrillation.

Etiology.—The stimulus for each contraction arises in the auriculo-ventricular node instead of in the sinus node. In the normal dog's heart the rate of rhythmic stimulus production in the auriculo-ventricular node is estimated by Eyster and Meek² to be about 67 per cent. that of the sinus node, and in the human heart the same proportion probably holds good. In health the sinus node is producing stimuli, to which the heart responds, at a higher rate than the auriculo-ventricular node, and consequently the functional activity

¹ Mackenzie, J., *Heart*, 1909-10, i., 23.

² Eyster, J. A. E., and Meek, W. J., *Arch. Int. Med.*, 1917, xix., 117.

of the latter is in abeyance and no contractions originate therein.

In animal experiments the dormant activity of the auriculo-ventricular node can be aroused (1) by stimulating it directly, for example by electric stimulation or by warming, or indirectly through the accelerator nerves, whereby its rate of stimulus production rises above that of the normal sinus node; and (2) by eliminating or depressing the activity of the sinus node (by excising, or by applying formalin or ethyl chloride to the sinus node; by its vagal inhibition, or by asphyxia) whereby the stimuli arising in the normal auriculo-ventricular node become effective. In all these experiments the auricles contract synchronously with the ventricles, the auricular deflexion in electrocardiograms is inverted, and, as the site of primary negativity is the auriculo-ventricular node, the whole heart is therefore responding to stimuli from this node.

If the activity of the sinus node has been eliminated or depressed, the rate of the whole heart is somewhat infrequent; if the rate of stimulus production in the auriculo-ventricular node has been exalted above that of the sinus node the cardiac rate is accelerated.

Clinically, the etiological factors are comparable to those causing nodal rhythm experimentally. We see cases of nodal rhythm with a cardiac rate that is approximately normal or somewhat retarded, in which the activity of the auriculo-ventricular node is revealed because of vagal inhibition of the sinus node; and the abnormal rhythm is abolished when the vagus is paralyzed by atropine. And we see others in which the cardiac rate is very frequent (from 130 to 230 per minute). When the acceleration recurs in paroxysmal form at intervals for many years it is apparently functional in origin.

In other cases the tachycardia is continuous. In a few cases of this nature the auriculo-ventricular node has been found to be the site of an acute inflammatory lesion which presumably caused it to discharge impulses at a high speed, and in many other patients the clinical and anatomical evidence strongly suggests the presence of a similar irritative lesion.

In a series of six of our cases¹ there was an acute endocarditis, which affected the aortic valve alone in one case, and the mitral, and sometimes the aortic and tricuspid valves as well, in the others. Four seemed to be due to a rheumatic infection, though the organism was only isolated in one case, and one to a streptococcal infection. In five of the cases a chronic endocarditis was also present. In all six cases the auriculo-ventricular node was involved in an inflammatory focus of recent date, and in four evidences of an old myocarditis co-existed. In five the bundle was also slightly involved, but the lesions there were always minimal.

In the case of malignant endocarditis reported by Falconer and Dean² an acute inflammatory lesion was found in the middle third of the bundle. Cellular infiltrations are frequently present in the tissues at the bases of inflamed valves, and from the close proximity of the mitral, tricuspid and aortic valves to the auriculo-ventricular node and bundle these latter are often involved, and characteristic disturbances of the cardiac rhythm make their appearance.

Nodal rhythm may, however, arise apart from endocarditis. In one of our cases, without any valvular lesion, the auriculo-ventricular node and bundle were involved in a subacute and diffuse myocarditis. Nodal rhythm has also been recorded in the course of rheumatism, enterica, scarlet fever and diphtheria. In these and in other acute infective diseases, inflammatory lesions often occur in the myocardium, and although they are most common in cases where endocarditis co-exists, they may occur when the valves as well as the pericardium are unaffected. The microscopic appearances of the essential lesions, the submiliary nodule, first demonstrated by Poynton³ in 1899, have already been described on page 48.

If the inflammatory lesion, whether it starts in the valves or in the myocardium, is an irritative one, a nodal rhythm, which may be preceded by nodal extrasystoles, develops. At first it may occur in paroxysmal form, alternating with a

¹ Cowan, J., Fleming, G. B., and Kennedy, A. M., *Trans. XVII Internat. Congress of Med.*, 1914, Sect. vi., Med. Part II, 223.

² Falconer, A. W., and Dean, G., *Heart*, 1912-13, iv., 137.

³ Poynton, F. J., *Lancet*, 1899, ii., 1163.

sinus rhythm over a period of several years ; or it may, when once developed, be continuous until death supervenes ; while in other instances nodal rhythm is the precursor of auricular flutter or fibrillation as terminal events. The patient seldom lives long enough for the lesion in the node or bundle to become destructive ; consequently auriculo-ventricular block is very rarely observed as a sequel to nodal rhythm.

Clinical Features.—Nodal rhythm is not a common disorder. Cases fall into one or other of three groups : (1) those in whom the cardiac rate is infrequent, or only slightly accelerated ; (2) those in whom it is persistently rapid ; and (3) those with paroxysms of tachycardia. The clinical features, however, depend not only on the rate of the heart and on the duration of the abnormal rhythm, but also on the inefficiency of the heart which necessarily ensues when the auricles and ventricles contract simultaneously, and on the extent to which the myocardium is involved by the inflammatory process.

1. NODAL RHYTHM WITHOUT TACHYCARDIA

This form of nodal rhythm, in which the immediate factor is vagal inhibition of the sinus node, is decidedly rare. F. N. Wilson¹ produced nodal rhythm in young persons by vagal stimulation following an injection of atropine, but before this drug had produced its maximal acceleration, and he advances the theory that this drug has a selective action on the vagus endings in the auriculo-ventricular node, paralysing them before it paralyses those in the sinus node.

The condition is also observed apart from the administration of atropine. For example, in a man aged forty-seven, suffering from subacute myocarditis without any valvular lesion, a sinus rhythm at a rate of about 90 per minute alternated during a period of one month with a nodal rhythm at a rate of about 80 per minute. The patient's condition was no worse when the latter rhythm prevailed, and it was revealed only by the characteristic records (Fig. 166) showing the abnormal form of each auricular deflexion, *P*. Sometimes the nodal rhythm lasted only for a few beats ; at other times

¹ Wilson, F. N., *Arch. of Int. Med.*, 1915, xvi., 989.

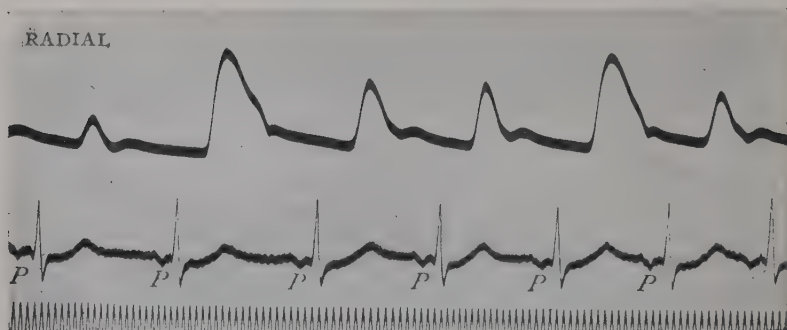


FIG. 166.—NODAL RHYTHM IN A MAN AGED FORTY-SEVEN.

Each auricular deflexion, *P*, is inverted, and the *P-R* interval is 0.10 second. There is slight irregularity of the ventricles, and the volume of the radial pulse waves is markedly irregular. Derivation II. 1.5 cm. = 1 millivolt.

it persisted for several minutes or hours. When it passed off it always did so gradually, the successive auricular con-

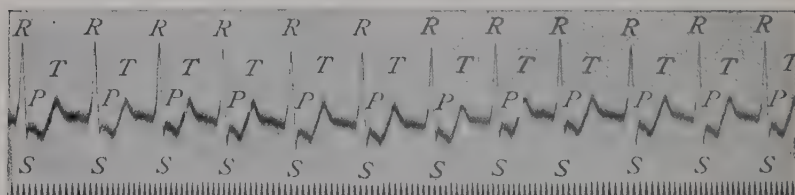


FIG. 167.—NODAL RHYTHM AT A RATE OF 71.4 PER MINUTE IN A WOMAN AGED FORTY-SIX. DERIVATION II.

tractions gradually regaining precedence of the ventricles until the normal auricle-ventricle sequence was regained. The patient made a transient recovery and resumed work.

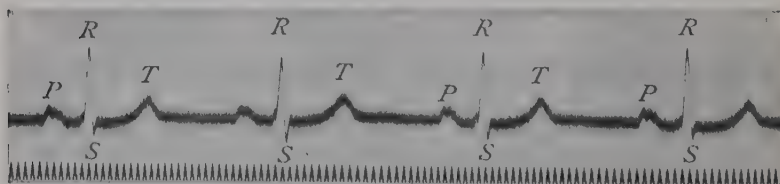


FIG. 168.—FROM THE SAME PATIENT AS FIG. 167, FOUR DAYS LATER, SHOWING RESTORATION OF THE SINUS RHYTHM AT A RATE OF 70 PER MINUTE. DERIVATION II.

But one month later he was re-admitted to hospital, cyanosed, breathless and dropsical; the auricles were then in fibrillation; he developed a hypostatic pneumonia and died three and a half months after he came under observation. The auriculo-

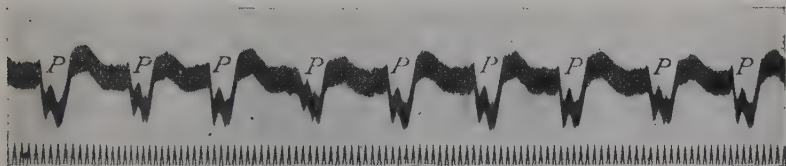


FIG. 169.—PAROXYSMAL VENTRICULAR TACHYCARDIA AT A RATE OF 134 PER MINUTE IN A CASE OF MALIGNANT ENDOCARDITIS. DERIVATION II.

ventricular node and bundle were found to be involved in a subacute myocarditis.

An infrequent nodal rhythm is, however, not necessarily attended by any signs of heart failure, and may not be detected unless graphic records are obtained. Thus Fig. 170 was obtained from a gamekeeper, aged fifty-five, a sparely built man

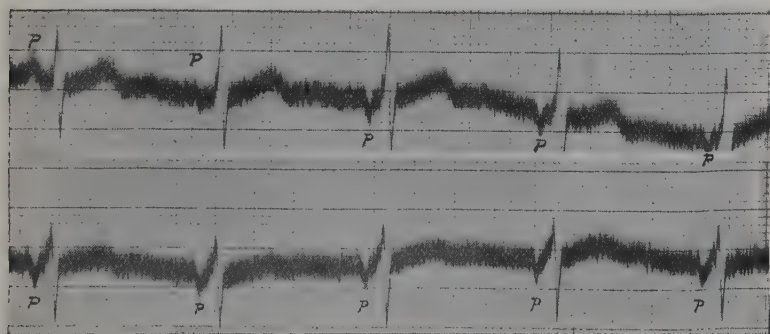


FIG. 170.—NODAL RHYTHM. MAN AGED FIFTY-FIVE. DERIVATIONS II AND III (see p. 203).

who looked younger than his years and could carry fourteen couples of rabbits on his back for a mile and a half without discomfort. The heart was of normal size, its sounds were pure, its rate was 75 per minute, and its rhythm regular. P_i was not seen, but P_{II} and P_{III} were inverted and the P - R interval was only 0.08 to 0.10 second. A fortnight later the nodal

rhythm was still present. A year later, when the man was still enjoying good health and when his organs appeared to be sound, a nodal rhythm was alternating with a sinus rhythm. The first three auricular deflexions, *P*, in the upper record by Derivation *II* of Fig. 170, show the change from sinus to nodal rhythm. The first beat, with a normal *P*, and a *P-R* interval of 0.14 second, is of sinus origin. In the second beat *P* is indistinct, but in the subsequent beats *P* is inverted, and the *P-R* interval is reduced to 0.10 second. The lower record, by Derivation *III*, in Fig. 170, represents a series of rhythmic nodal beats, each with an inverted *P* and a short *P-R* interval. At the onset of the nodal rhythm in this case the rate of the heart did not alter appreciably, diastole only shortening 0.01 to 0.02 second. Three years later the man was in excellent

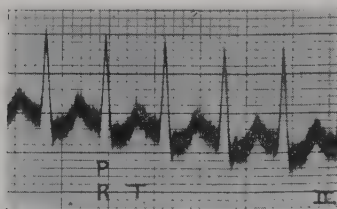


FIG. 171.—PAROXYSMAL NODAL TACHYCARDIA.

Rate is 232 per minute. Man aged sixty; systolic blood pressure, 200 mm. Hg. He died three years later. Derivation *II*.

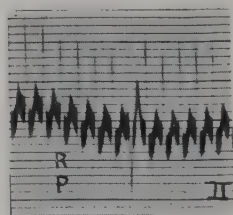


FIG. 172.—PAROXYSMAL NODAL TACHYCARDIA AT A RATE OF 232 PER MINUTE.

A ventricular extrasystole occurs after the seventh beat. Derivation *II*.

health, working steadily as an estate forester; the left heart was slightly enlarged; its rate was 71; the sounds were pure, the blood-pressure was 100–125 mm. Hg., and the electrocardiograms were normal.

This case is of interest because nodal rhythm, and especially that form in which the pulse is not accelerated, is not common. In one of the cases recorded by Heard and Strauss,¹ a nodal rhythm, with *P* succeeding *R*, recorded during ether anaesthesia, the pulse rate was about 92; in one of Lewis's² cases

¹ Heard, J. D., and Strauss, A. E., *Amer. Journ. Med. Sci.*, 1918, N.S., clv., 238.

² Lewis, T., *Quart. Journ. of Med.*, 1912–13, vi., 221.

the pulse rate was 48, and similar cases have been recorded by Laslett¹ and by one of us.² In Mathewson's³ case a nodal rhythm with a rate of about 63 was frequently alternating with a sinus rhythm having a rate of about 68. In this case sympathetic stimulation or physical exertion lead to the temporary abolition of the abnormal rhythm.

In cases such as these the inception of the nodal rhythm produces no particular symptoms, and the patient is usually unconscious of the disorder. The only symptom of which he is apt to complain is palpitation, which is then directly due to the synchronous contraction of auricles and ventricles. Atropine may abolish the nodal rhythm and its resultant palpitation.⁴ In exceptional cases the cardiac rate is markedly retarded, the disorder being then termed nodal bradycardia.

2. CONTINUOUS NODAL TACHYCARDIA

This form of nodal rhythm is occasionally detected in the course of a detailed examination of a patient who is suffering from acute endocarditis, rheumatic fever, diphtheria⁵ or other acute infective disease, or who is suffering from terminal heart failure consequent upon mitral or aortic disease. In many cases the patient's condition is already so critical that the development of a nodal rhythm adds little to its gravity. But the synchronous contraction of auricles and of ventricles always impairs the heart's efficiency, the acceleration of rate may in itself hasten the onset of cardiac failure, and the inception of the new rhythm is sometimes the signal for the onset of urgent symptoms.

This form of nodal rhythm sometimes occurs as a terminal event in acute endocarditis.

One of our cases, a man aged forty-two, had been ailing for nine months. He had to sit bolt upright in bed, coughing continually and expectorating large quantities of watery mucoid material. Œdema

¹ Laslett, E. E., *Heart*, 1915-17, vi., 81.

² Ritchie, W. T., *Auricular Flutter*, Edin. and Lond., 1914, 118, 120.

³ Mathewson, G. D., *Quart. Journ. of Med.*, 1915-16, ix., 1.

⁴ Bishop, L. F., *Journ. Amer. Med. Assoc.*, 1921, lxxvii., 31.

⁵ Hume, W. E., *Heart*, 1913-14, v., 25.

was universal, and very marked in the legs and scrotum. The heart was enlarged, a double aortic murmur was audible over the sternum, and evidence was found of a consolidation of the middle lobe of the right lung and of pleurisy on the same side. Frequent nodal extrasystoles occurred, either singly or in series of 2, 3, or 4, during the first four days after his admission to hospital. On the fifth day the pulse was regular, with a rate of 110 to 120, and distinctly alternating; tracings showed a nodal rhythm, with an *a-c* interval as short as 0.1 second. He died suddenly on the sixth day. All the aortic cusps showed small, recent, warty vegetations, but were not thickened, and one cusp was incompetent, being perforated a little below its free border. A small abscess, as well as a large infarct, were found in the middle lobe of the right lung. The auriculo-ventricular node was involved in an acute inflammatory process, which was most marked in the upper portion of the node and in parts which were nearest to the base of the mitral valve. The sinus node was normal. The cardiac muscle throughout showed evidence of cloudy swelling and a few inflammatory nodules, which were most numerous at the base of the mitral valve and in the auricular septum just above the node.

Another patient, a girl aged ten, had been ailing for two months before she became short of breath and complained of pain in the abdomen and beneath the sternum when she coughed. On admission to hospital the child looked ill and exhausted and was inclined to be drowsy. The face was pallid and puffy, but there was no oedema. Both sides of the heart were enlarged; a long soft blowing murmur was audible at the apex, while the second sound was reduplicated. The nature of the infection was obscure, the joints were never swollen or painful, and the tonsils, though large, were not acutely inflamed. She was habitually fevered at night, but the temperature only once rose above 101° F., and was usually under 100° F. The leucocyte counts varied between 8,000 and 23,000. No focus of infection was discovered, and the symptoms pointed to a rheumatic infection of the heart without arthritis. An urticarial rash appeared a fortnight later. This was followed by pericardial friction and a small pleural effusion at the base of the left lung; a crop of minute subcutaneous nodules appeared on the backs of the hands; exhaustion became more extreme; and she died six weeks after admission to hospital. The heart maintained its sinus rhythm, with an occasional ventricular extrasystole, until the last week of life, when a nodal rhythm with a rate of about 133 per minute was recorded.

Microscopic examination of the heart revealed a well-marked interstitial myocarditis. The nodules were most abundant in the neighbourhood of the inflamed mitral and tricuspid valves. A large inflammatory focus near the tricuspid valve infiltrated the bundle for a short distance. The auriculo-ventricular node was notably affected; its capillaries were congested, and the lymphatic vessels were plugged with masses of lymphocytes. The principal vein of the node showed an acute phlebitis and peri-phlebitis.

A continuous nodal tachycardia was observed for a month before death in the following case. The patient, a woman aged twenty-seven, had suffered from recurrent attacks of acute arthritis with fever for two months. This was associated with a leucocytosis varying from 16,000 to 26,000. The only visceral abnormality at first was a mitral lesion, but the repeated recurrence of arthritis, and the occurrence of pericarditis and subcutaneous nodules indicated a persistent rheumatic infection. Blood cultures were sterile. The heart had a sinus rhythm, she made a satisfactory recovery, continued in good health for a year after leaving hospital, and made a good recovery from a confinement. Four months later she was re-admitted to hospital, seriously ill, with arthritis and pneumonia, the temperature was usually remittent, ranging from 100 to 102° F., the pulse was always regular at a rate between 110 and 120 and reaching 140 when the temperature was high. For one month the heart maintained its sinus rhythm, but for one month before death there was a nodal rhythm with an *a-c* interval measuring 0.10 second, or less. An extensive fibrosis was found spreading from the mitral valve into the auriculo-ventricular node, in which there were many fibroblasts and round cells. The cellular infiltration spread above and beyond the node into the auricular muscle, but the bundle, the sinus node and the vagus nerves were normal.

3. PAROXYSMAL NODAL TACHYCARDIA

The occurrence of paroxysms of rapid cardiac action was first described by Cotton in 1867. It is now known that in many instances these paroxysms are essentially a greatly accelerated nodal rhythm,¹ while others are known to be auricular flutter or more rarely paroxysmal auricular fibrillation, or to be ventricular in origin.

Paroxysms of nodal tachycardia are markedly similar even in different patients. The onset is sudden and apparently instantaneous, and the termination is equally abrupt. An attack may commence without obvious cause, but is frequently and especially in the earlier phases the sequel to physical exertion. As time goes on the degree of exertion required often diminishes, so that attacks ensue on apparently trivial physical strain. Sometimes they follow straining at stool. In a patient under our care they at first succeeded severe fits of coughing, during which the patient became extremely livid, her face swelling rapidly to nearly double its usual size, and the jugular veins becoming distended to three or four

¹ Rihl, J., *Deutsch. med. Wochenschr.*, 1907, xxxiii., 632. Lewis, T., *Heart*, 1909-10, i., 306.

times their usual diameter. The pulse became extremely frequent and uncountable, and lost markedly in volume and strength, and continued in this way for several minutes, when the infrequent rate was rapidly resumed.

Sometimes the paroxysms succeed mental excitement, and, in consequence, they not infrequently occur in the consulting-room, or at demonstration in the wards. In one of our patients, who required operation on account of acute appendicitis, the frequency of the pulse rate excited apprehension of widespread peritoneal involvement, which proved, however, to be unfounded, and the pulse rapidly resumed its normal rate after operation. Flatulent distension of the stomach, too, may be an exciting cause.

The cardiac phenomena are characteristic. The pulse rate is greatly increased, and numbers 150 and upwards; 160 to 180 seem to be common figures, but we have records of 226 and 234 beats per minute, and even higher figures have been reported, the maximum being about 300. Coincidentally the pulse loses both in force and in volume, and becomes small, soft and quick, irrespective of its previous character. The cardiac pulsations, too, become exaggerated, pulsation being noticeable in several interspaces on both sides of the sternum, though diffuse and badly sustained. The whole chest may shake, and the vehemence of the heart's contractions contrasts in a striking way with the smallness and softness of the pulse.

The symptoms of the patient depend upon the amount of the cardiac reserve. In uncomplicated cases they are often surprisingly slight, and the consciousness of the heart-beats may be the sole subjective sensation.

For example, in one of our patients, an iron-dresser aged forty-five, twenty-one attacks were observed in the course of fourteen weeks. They commenced suddenly and without apparent cause, and occurred at any period of the twenty-four hours, during sleep as well as at other times. The shortest lasted for but a few minutes, the longest for forty-two hours. At the onset the pulse rate changed abruptly from 60 or 64 to a rate varying from 134 to 180. During the paroxysms the heart was always perfectly regular, the pulse was very quick and soft, and the systolic pressure fell to 85 mm. Hg. during the four attacks in which it was recorded, as contrasted with 115 to 125 mm. Hg. in the intervals. The termination of the attacks was as abrupt as their commencement,

the normal rate instantly succeeding the rapid action. He was but slightly incommoded during the attacks, and though he usually appreciated their commencement and end, and the palpitation during their continuance, he was not always conscious of the tachycardia, and was accustomed to feel his pulse to ascertain what the rate was. He had no dyspnoea, orthopnoea, cyanosis or oedema during the attacks; his skin, however, was congested, and he sweated fairly freely, but he took his food and went to sleep as usual during a paroxysm, and would probably have walked about save that he was confined to bed by order.

For nearly two years after his discharge from hospital he maintained fair health, and continued his work as an iron-dresser, though short attacks of palpitation recurred about every fortnight. One attack lasted for two days, and he was off work for a week. Three months later an attack began and continued without interruption for four days, when he was re-admitted to hospital. This paroxysm was preceded by abdominal pain, was accompanied by nausea and retching, and subsided after a bout of vomiting, after which the pain disappeared. He remained in hospital for nearly a month, and had only a few paroxysms, the longest of which lasted for twelve hours. The highest pulse rate recorded was 180; his general condition was good; and no change was found in the viscera.

Another patient, a woman aged thirty-three, had suffered from paroxysms of tachycardia for eight years, each being preceded by nausea and sickness. During the paroxysms, which started abruptly, the ventricular rate was 168 to 226 per minute, the arterial pulse became almost imperceptible, but the pulsation of the jugular veins was large and forcible, and the liver was felt to pulsate freely. Vagus compression did not arrest the paroxysms.

In long-continued paroxysms, evidence of venous stasis may be observed—breathlessness, congestion of the liver, albuminuria, a scanty concentrated urine, and oedema of the extremities.

In patients who are already seriously ill the occurrence of the tachycardia rapidly augments the distress.

In a labourer, aged thirty-eight, the earliest symptom was the occurrence of attacks of palpitation, which were generally nocturnal. At first they occurred about once a week and lasted from thirty minutes to three hours; but they gradually became more frequent and of longer duration. On admission to hospital he was extremely ill, in full orthopnoea, the respirations being laboured and cyclic. The heart was greatly enlarged, and a double aortic murmur was audible; the liver was enlarged; the urine contained much albumin; the pulse was regular and shotty and numbered 88 per minute. In ten days improvement was marked, and he was breathing easily and lying low in

bed. The liver was smaller and the albuminuria was trifling. Four days later, however, a paroxysm of tachycardia ensued and lasted for eight hours, the pulse rate varying between 152 and 180, and all the former symptoms at once recurred. Next day he was much better, but a few days later another attack of tachycardia supervened, and though it only lasted for an hour, notably intensified his distress. The pulse rate, too, only fell to 110 after the attack had passed off. In the evening another paroxysm ensued, the pulse rate numbering about 200. The cyanosis became extreme and the dyspnœa urgent, and after three hours the heart suddenly ceased to beat.

On examination the heart was very large, weighing $25\frac{1}{2}$ ounces. The aortic valve was notably incompetent from chronic sclerotic changes, the aorta was very atheromatous, and the orifices of the coronary arteries were considerably narrowed. The kidneys showed widespread fibroid change.

In this case the tachycardia seemed directly responsible for the fatal termination, as improvement had been considerable after his admission to hospital. The post-mortem examination, however, showed widespread valvular and myocardial disease, so that the cardiac reserve must have been small.

A similar fatal issue was observed in a big stout woman aged fifty-three, who was suffering from chronic bronchitis and emphysema. The paroxysms of tachycardia were closely related to bouts of coughing, and at first they ceased shortly after the cough; but later they persisted indefinitely and rapidly produced fatal cardiac failure. Four days before death the cardiac rate in a paroxysm was 222. A paroxysm with a rate of 234 recurred two days before death, and persisted until the end.

The coincidence of the auricular and ventricular contractions explains several of the characteristic features of the paroxysms. During ventricular systole the auriculo-ventricular valves are shut, and the auricle contracting simultaneously with the ventricle is, in consequence, unable to supply the latter with its normal quantity of blood. The ventricular output is thus lessened and the pulse loses both in volume and in force.

The auricular contraction drives the blood backward into the veins, and thus initiates venous stasis. If the paroxysm is prolonged, the degree of stasis rapidly becomes serious, the lungs and liver becoming engorged and œdema making its appearance in the dependent parts. The pulsations in the neck, too, become larger, more distinct and forcible, and the liver pulsates freely.

The obstacle to the output from the auricles stimulates

them to increased effort, and the pulsations on the front of the chest become more forcible and more widespread, and the apparent strength of the cardiac contractions contrasts markedly with the smallness and softness of the arterial pulse. The cardiac sounds acquire the foetal rhythm, and if there be a pre-existing mitral systolic murmur it becomes enfeebled or disappears because mitral regurgitation is lessened or abolished by the auricles being in contraction synchronously with the ventricles.

Tracings from the jugular vein show one large wave commencing at the beginning of ventricular systole and ending when the tricuspid valve opens ; or if the *a* wave precedes *c* the *a-c* interval is reduced to about 0.10 second. If the end of a paroxysm is recorded graphically, there is seen to be a post-paroxysmal pause, comparable to a post-extrasystolic pause, as described on page 88, before the first contraction of sinus origin occurs. In the intervals between paroxysms the jugular pulse is of normal form, and the *a-c* interval is no longer shortened.

Electrocardiograms vary somewhat in different cases. In some an inverted auricular deflexion is seen before each ventricular complex, but the auriculo-ventricular (*P-R*) interval is only 0.10 second, whereas in health it is 0.14 second. In others the auricular deflexion, occurring during the ventricular complex, is recognizable only by the abnormal notching of the latter, or *P* may be buried in *R* (Fig. 171).

The Diagnosis of paroxysmal nodal tachycardia depends mainly on the extreme speed of the heart, the paroxysmal nature of the attacks, their abrupt onset and ending, and the perfect regularity of the pulse during the attacks.

The sudden onset, the rapidity of the pulse at a rate exceeding as a rule 140 and sometimes exceeding 200 per minute, the absence of any respiratory irregularity of the pulse, and the abrupt termination of the paroxysm differentiate it from a sinus tachycardia. The regularity of the pulse excludes auricular fibrillation ; the large forcible jugular pulsation at the same rate as the arterial pulse, and the inability to retard the ventricles by compression of the vagus, exclude auricular flutter.

Paroxysmal Ventricular Tachycardia,¹ in which each contraction is essentially a ventricular extrasystole, is one of the rarest forms of paroxysmal tachycardia. The cardiac rate may attain a speed of 260 per minute, and the venous pulse is of the ventricular form as in nodal tachycardia. The paroxysms seldom last for more than a few minutes, but may last for some hours or for several days.² The condition is depicted in Fig. 169, taken from a man, aged forty-one, five days before death from malignant endocarditis of the aortic and mitral valves. The heart was usually beating with a sinus rhythm, but sometimes there was a coupled rhythm owing to regularly recurring ventricular extrasystoles. Fig. 169 is a portion of a long series of ventricular extrasystoles at a rate of 134 per minute, and each complex is notched by an auricular deflexion, *P*. The auricular rate may be only half that of the ventricles if the latter is about 200 a minute. The rate of the ventricles is unaffected by atropine, and the ventricles are not retarded by compression of the vagi.

The Prognosis in paroxysmal tachycardia must be based upon several factors. Paroxysms of short duration and infrequent occurrence, which have never produced any signs of cardiac insufficiency, occurring in patients with no obvious diminution in the "field of response," are of little practical importance, save that they indicate a tendency to functional disturbance which may in time prove serious. Herringham³ states that the condition seems always to get worse and in the end to kill, and that after thirty the patient is never safe, though the progress may be slow, since people who have been affected in childhood have been known to pass fifty years. One of our patients has suffered from attacks for more than forty-five years, and is still hale and fairly active. On the other hand, paroxysms of considerable duration and frequency, producing signs of obvious cardiac distress and occurring in patients whose hearts in the intervals exhibit little cardiac reserve, cause extreme danger. A paroxysm which lasts

¹ Hart, T. Stuart, *Heart*, 1912-13, iv., 128. Hume, W. E., *Quart. Journ. of Med.*, 1917-18, xi., 131.

² Robinson, G. Canby, and Hermann, G. R., *Heart*, 1921-22, viii., 59.

³ Herringham, W. P., *Edin. Med. Journ.*, 1897, N.S., i., 366.

for two or three days seems always to produce symptoms, and the duration of the paroxysms is probably of most importance in this respect.

The Treatment of the paroxysms is still unsatisfactory. The vast majority of attacks end spontaneously, and the patient is restored to his usual health, but there is little that can be done to induce the cessation of the frequent action. We have seen attacks cease after deep breathing, holding the breath, exertion, the act of vomiting, the application of fomentations to the præcordia, and they have been described as terminating after pressure had been exercised upon the chest or upon the vagus nerve in the neck. But every one is agreed that those measures usually fail, and that their effects are rarely uniform even in the same patient. Digitalis, too, has proved equally useless in our hands, although good results have been recorded after its administration.

Much, however, can be done in the intervals to lessen the frequency of the attacks. A careful and detailed survey of the case usually shows some manifest error in the mode of life, some functional disturbance in one or other of the viscera, or some focus of infection in the tonsils or elsewhere. As Herringham points out, over-indulgence in tea, coffee, alcohol, or tobacco, or some digestive disturbance, are sometimes responsible. In one of our patients a gastro-intestinal intoxication accompanied by obstinate constipation is apparently correlated to the frequency of his attacks. The cardiac reserve may be limited even in the absence of organic valvular or myocardial disease, and the usual modes of treatment are necessarily indicated. Or the cares of business or domestic worries may press unduly upon the patient.

It may appear unnecessary to limit the physical exercise of these patients if, when seen between attacks, their general health and nutrition seem unimpaired, but our personal experience has taught us caution in this respect. A paroxysm *in esse* is always a period of danger, for it is impossible to predict its duration, and if it continues for more than a day or two, definite signs of cardiac failure are sure to appear. The heart, too, is unduly irritable, whatever the source of this may be, and an irritable organ is hypersensitive to strain and stress,

so that it is generally advisable to exercise precautions in this direction. If the attacks are frequent, however short their duration, or if any evidence of cardiac insufficiency is present, the patient should be confined for a time to bed. In the milder cases less restriction is indicated. The obvious errors should at the same time be corrected, special attention being paid to the alimentary tract, and a general tonic may be added. Small doses of digitalis seem in many cases to help.

CHAPTER XIII

ACUTE ENDOCARDITIS

THE clinical distinction between simple and malignant forms of acute endocarditis is apt to convey an erroneous idea of their true nature. Both are due to bacterial invasion, and the pathological difference is merely a matter of degree, the infection in the one case being readily overcome, and in the other progressing indefinitely; and an intermediate group connects the links at each end of the chain. The difference may be due to several causes: to the particular variety of bacterium, to its virulence or its dose, or to the resistance of the tissues to the infection.

The distinction, too, is inaccurate from the clinical standpoint. As Sir William Osler said, there is no *benign* endocarditis, for even the most trivial acute infection may inaugurate changes of a chronic character, which sooner or later lead to death; while an infection which is at first trivial in its grade may suddenly assume a virulent character and rapidly produce a fatal issue. The malignant cases, too, may change their character, and the fire which blazed brightly at first may ultimately burn itself out.

The Lesions.—In the majority of cases acute endocarditis involves the valves alone, and does not affect the endocardial lining of the auricles and ventricles. In an early case the characteristic festoons of little translucent white or yellow beads are visible running round the cusps, a short distance from the free margins, at the points where the contact is most intimate when the valves are shut (Fig. 173). The surface which is exposed to the blood-stream, namely the ventricular aspect of the arterial valves and the auricular aspect of the auriculo-ventricular valves, is alone implicated. The mitral

and aortic cusps, which are exposed to the highest blood-pressure and bathed in the most highly oxygenated blood, are more frequently affected than the valves on the right side of the heart. Microscopic examination shows that the vege-



FIG. 173.—ACUTE VERRUCOSE ENDOCARDITIS, FOLLOWING A RHEUMATIC (OR GONOCOCCIC) ARTHRITIS (WESTERN INFIRMARY MUSEUM).

tations are mainly composed of fibrin, with a few cells scattered through the mass, and a larger number along the free margins. At the base the connective tissue of the valve is hyperæmic and proliferating, and a cellular infiltration may be found.

Micro-organisms may be detected with suitable staining, and are, as a rule, most numerous in the superficial layers, though they are sometimes seen in the deeper layers and in the valve itself.

The infection of the valves may arise from the blood stream in, at any rate, aortic endocarditis, for in the healthy adult valve blood vessels are rarely present. The bacteria gain access to the tissues through some abrasion of the protective endothelium produced by the toxæmia and the mechanical stress imposed upon the parts. The mitral valve, however, frequently contains vessels, and endocarditis may follow bacterial embolism of the deeper structures, the exudate forming when the inflammation reaches the surface, and the endothelium is shed.

The researches of Gross¹ upon the blood supply of the heart make it probable that the second cause is the most common. In the foetal heart the valves contain a considerable amount of muscle, well supplied with blood vessels, but, as age increases, both muscle and vessels rapidly lessen in amount, and in the adult, though the exceptional heart may still show a few vessels, the large majority show none. As the incidence of endocarditis from the age point of view is the same as the incidence of the presence of vessels in the valves, embolism would appear to be the most common cause of acute endocarditis.

In a case of endocarditis which was shown to us by Kennedy, the infection arose in an unusual way. An abscess had formed in the inter-ventricular septum, and reached the interior of the right ventricle immediately beneath the medial cusp of the tricuspid valve. The latter became involved by direct infection, and a perforation measuring nearly 1 cm. in diameter ensued in the centre of the cusp. The free margin was not affected.

In some cases no bacteria are found on microscopic examination and the reaction in the valve is minimal. This is the general rule in the endocarditis of acute rheumatism, but, although the lesion might follow a toxic and traumatic damage to the valve, the general evidence in favour of an infective origin of acute rheumatism is so strong that one is forced to

¹ Gross, L., *The Blood Supply to the Heart*. New York, 1921.

the conclusion that the cardiac condition is due to a similar infection of the valve. In the majority of cases of this type the inflammation in time subsides. The vegetations are penetrated by new-formed connective tissue, and the surround-

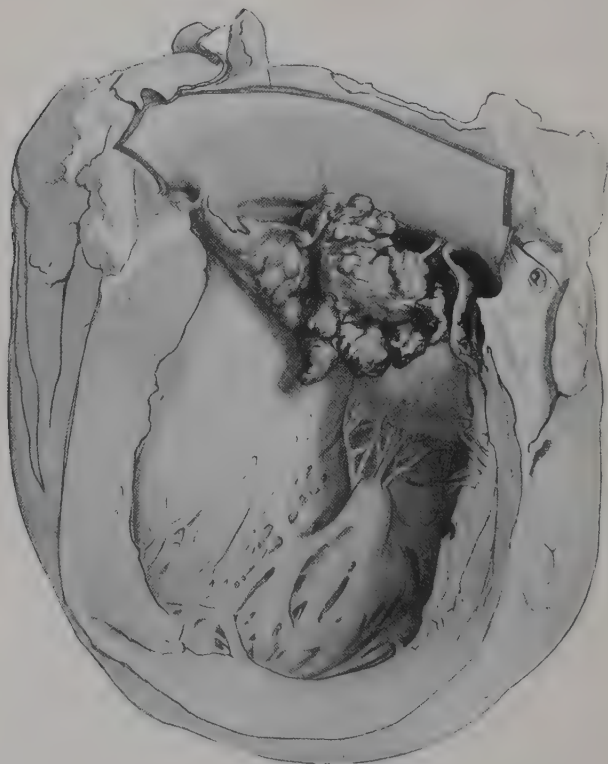


FIG. 174.—PNEUMOCOCCIC ENDOCARDITIS OF AORTIC VALVE SPREADING ON TO THE MITRAL CUSPS (WESTERN INFIRMARY MUSEUM).

Embolic gangrene of the foot ensued ten days after the onset of the pneumonia.

ing endothelium grows over the denuded surface, leaving a thickened scar of greater or of less extent.

In the malignant type of case the ultimate result is very different, the bacterial infection is not overcome, and the infection spreads. In some cases the vegetations are large and luxuriant, measuring many millimetres in length (Fig. 174) and interfering by their mere bulk with the proper action

of the valves. In others the process is destructive, gross necrotic and ulcerative processes ensue, and the cusps may be perforated (Fig. 175), or their attachments may be severed,

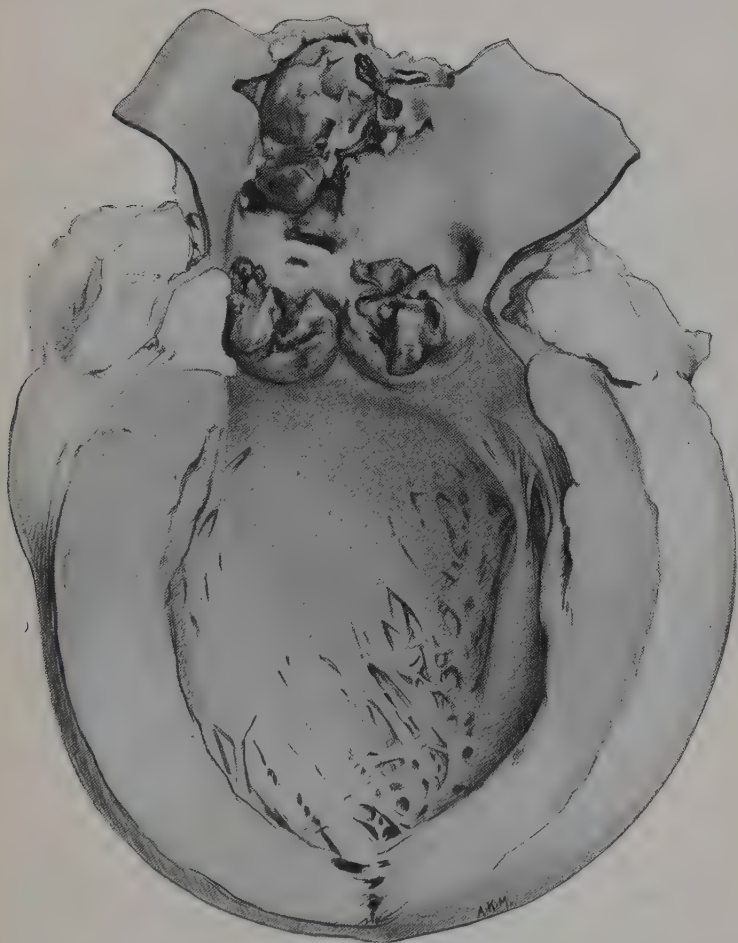


FIG. 175.—MALIGNANT ENDOCARDITIS OF AORTIC VALVE, WITH ANEURYSMS AND PERFORATIONS OF THE CUSPS; INFECTIVE AORTITIS; AND ANEURYSM OF AORTA RUPTURING INTO PERICARDIAL SAC (GLASGOW ROYAL INFIRMARY MUSEUM).

and their function may be almost wholly abolished. The inflammation too, in some cases, spreads from the valves to the adjacent parts. The spread is often direct, and takes

place in any direction. If the aortic valve is involved, the aorta may be affected, and an acute infective aortitis results, with sometimes aneurysmal dilatation (Figs. 175, 176); or the inter-ventricular septum may be infected (mural endo-



FIG. 176.—PNEUMOCOCCIC ENDOCARDITIS OF THE AORTIC VALVE (WESTERN INFIRMARY MUSEUM).

One cusp is partly severed from its attachments. An aneurysm, the size of a walnut, sprang from the aorta immediately above the valve, and projected into the right auricle.

carditis), and the ulceration may spread deeply into the tissues, and even open up communication with the right side of the heart. When the mitral valve is implicated, either the auricle or ventricle may be invaded. The most common invasions are upwards on to the auricular wall opposite to the

foramen ovale (contra-fossal wall) (Fig. 177), and downwards on to the chordæ tendineæ (which may be ruptured in the process), sometimes invading even the tips of the muscoli papillares.

In other cases the infected area is situated a little distance



FIG. 177.—STREPTOCOCCIC ENDOCARDITIS OF MITRAL VALVE SPREADING ON TO THE LEFT AURICULAR WALL (WESTERN INFIRMARY MUSEUM).

The chordæ tendineæ are also involved.

from the valve, from an infected “tag” waving to and fro in the blood-stream, and hitting and infecting the ventricular, auricular, or aortic walls. Or a fragment of a vegetation may be broken off, enter a coronary artery, and produce an embolic abscess anywhere in the heart.

The infective character of malignant cases is always well

marked, and large collections of bacteria may be seen on the surface of the vegetations, or more deeply situated in the valve itself.

In the intermediate cases the lesions are less extreme and more chronic in their character. The general evidences of septicæmia may be absent or trifling, but a progressive valvular lesion can be detected on physical examination. In the valves themselves progressive and reparative changes may be seen side by side, but the valvular defect is always severe and the patient often dies from the cardiac fault even when healing has taken place.

It seems certain that in the mildest cases a complete recovery may occur, but pathological data are necessarily absent. In the majority of cases, though the infection is overcome, a scar is left behind. The cardiac valves have, however, no rest, by day or night, and chronic fibrous changes ensue, the cusps becoming thickened and shrunken and often adherent to each other, and the valvular defect gradually but steadily increases in degree. In time, after perhaps many years, it becomes extreme, and the cardiac "pump" fails to accomplish its necessary work.

In acute endocarditis the *cardiac muscle is invariably damaged* to a greater or a less degree. The gross infective and ulcerative lesions which occasionally occur in malignant cases have, of course, for long attracted notice, but the microscopic lesions have failed to receive their due share of attention, though myocarditis is an almost constant accompaniment of acute endocarditis (*see pp. 48, 366*). The lesions are usually most extreme in the vicinity of the base of the affected valves, but they are often disseminated widely through the muscle. Their degree does not necessarily correspond with that of the valvular inflammation, and they may be well marked in cases where the valvulitis is but trivial. In some cases clinical evidence of an acute myocarditis is available, for the auriculo-ventricular node and bundle are in close proximity to the mitral, aortic, and tricuspid valves, and may be involved in the inflammation; and heart-block, extrasystoles, or nodal rhythm may, in consequence, develop.

Pericarditis is a frequent complication. The flow of blood and lymph takes place towards the pericardium, and its

infection is of common occurrence, and necessarily adds greatly to the gravity of the case.

The Causes of Acute Endocarditis.—(1) **Acute Simple Endocarditis** occurs most frequently in childhood and in adolescence, though it may be found at any period of life ; but accurate statistics are not available, for it rarely causes death during the acute stage, and *post-mortem* data are, in consequence, wanting. This predominance is chiefly due to its close connection with *acute rheumatism*—a connection which has been amply proved by clinical observation. In a series of 1,750 cases collected from the literature, 933 patients suffered from endocarditis ; and its incidence is twice as great in persons under twenty years of age as it is in older persons. In children under ten, endocarditis and acute rheumatism are almost always conjoined, and Norman Moore aptly named the disease *cardiac fever*. Endocarditis is said to occur most frequently in the first attack of acute rheumatism, and within the first fortnight of the fever ; and its incidence is not directly related to the severity of the arthritis, as it is of common occurrence in cases where the arthritis is trifling or subacute, and may be absent though the arthritis is severe and prolonged. A recent “claim” in an insurance office illustrates the point. The man died at the age of fifty-eight from mitral stenosis. He had had muscular rheumatism in 1874, and rheumatic pains in his joints, “not rheumatic fever,” for two or three months in 1884.

The mitral valve is most frequently affected, and the valves of the right heart are but seldom involved.

Endocarditis is frequently associated with *chorea*, and was present in 154 cases of a series of 171 examined *post-mortem*. Valvular lesions are extremely common in choreic patients, and were present in 262 out of 829 patients who were examined during, or after, the illness. The endocarditis is usually of the simple type, and though so often present in fatal cases, is rarely the immediate cause of death.

In association with the *exanthemata* endocarditis is comparatively rare, but here again *post-mortem* data are misleading, as in some diseases endocarditis is apt to be of the malignant type, and an active factor in producing death ;

and its relative importance is, in consequence, over-estimated. In *pneumonia*, for example, it was found *post-mortem* in 16 out of 100 cases (Osler), but its clinical incidence is slight (0.44 per cent., Musser and Norris). In *scarlatina* it only occurred in 0.65 per cent. (McCollum). Macrae reported its occurrence in 6 of 1,500 cases of *enteric fever*, and Horton-Smith in 4 of 290 cases examined *post-mortem*; and Councilman in 1 out of 54 (*post-mortem*) cases of *small-pox*. Horder has recorded its occurrence in *influenza*, in which disease it is not infrequent.

The causes of acute endocarditis, with the exception of acute rheumatism and chorea, are thus ill defined, and it has been suggested that the other "rheumatic" manifestations, acute tonsillitis, erythema nodosum, etc., may be responsible. The suggestion is extremely plausible, for the tonsils are recognized to be the harbourers of many micro-organisms, and not infrequently show on section macroscopic evidence of gross infection of the crypts which was not evident on examination of the surface. The probability of a blood-infection is increased if the middle ears and the nasopharynx are, as is so frequently the case, also inflamed.

It must be remembered, however, that the portal of entry in malignant cases is not always evident, even on *post-mortem* examination, and in these cases must have been trivial and of short duration; and a similar trivial sepsis may be responsible for some of the simple cases.

(2) In the **malignant** cases the valves which are involved have been as a rule already damaged, and are, in consequence, places of lessened resistance, so that the disease affects older people than in the case of the simple form. In Kanthack and Tickell's series of 84 cases, only 25 were under twenty years of age, and in Horder's series of 150, only 45. In our series the proportion is larger, 25 out of 70 cases. The valves had been previously damaged in 64 per cent. of Kanthack's and 80 per cent. of Kelynack's series; in 90 per cent. of Horder's series; and in 62 per cent. of our series. Malignant cases are, however, not uncommon in the second decade, perhaps in consequence of the special incidence of acute rheumatism at this period.

The association with acute rheumatism is again extremely close, but the nature of the connection is not clear. Rheumatism may act merely as a predisposing cause, damaging the valves and so permitting a subsequent bacterial infection ; or by producing a malignant form of endocarditis. Poynton has emphasized the latter view and has isolated the micrococcus rheumaticus in blood-culture on several occasions. In one of our cases Kennedy isolated a similar organism both during life and after death. But Horder considers that malignant rheumatic cases are due to a superadded infection. In the typical endocarditis of acute rheumatism, at any rate, blood-cultures are almost always sterile.

The association with other infections is by no means close. The majority of these patients have, of course, suffered from some of the exanthemata in early life, or from some infection, but in a few cases no antecedent illness has been known. In others, however, many infections have been present, and one of our patients, a man of twenty-four years, had suffered from growing-pains, enteric fever, diphtheria, pneumonia, and gonorrhœa ; he was liable to boils, and his mouth was extremely septic, the gums being swollen and bleeding on slight provocation. A similar state of extreme oral sepsis was present in another patient, while a third had an offensive rhinitis, and a fourth a chronic otorrhœa. In two of our cases the symptoms developed during lactation, though there was no obvious source of sepsis about the uterus or breasts.

In another case the symptoms succeeded the birth of the patient's second child. She had frequent rigors, with profuse perspiration, which recurred every two or three days. The lochia persisted for six weeks, and only finally cleared after "a copious discharge" had continued for a few days.

In another the symptoms succeeded a "cross-birth" twelve weeks before admission to hospital. Rigors occurred on the third or fourth day after her admission, and were frequently repeated, and she felt pain in her left side when she coughed. *Post-mortem* examination revealed the association. The uterus was small and involuted, but the endometrium showed evidence of recent ulceration, and many thrombosed veins were present

in the muscular wall. The right ovarian vein contained a long thrombus, and there were clots in the iliac veins and the lower three inches of the inferior vena cava; these did not, however, completely block the lumen. There was a large, dense, mobile thrombus, the size of a marble, in the right auricle, and a smaller one attached to the wall. The mitral valve was covered with fairly large, soft, fibrinous vegetations, and the tricuspid valve was also affected, but in lesser degree. Acute abscesses were present in the spleen and in the lungs, and a large slough of the visceral pleura had produced a pneumothorax, which was the immediate cause of death. Blood-cultures during life were sterile.

Hearts with congenital defects are not infrequently affected.

The valves on the left side of the heart are those which are most frequently involved, but those on the right side of the heart are more often involved than in the simple form. The mitral valve is that most frequently infected. Two or more valves are involved in nearly half the cases, and a mural infection occurs in about a quarter.

Recent improvements in blood-culture have led to the isolation, during life, of the causal micro-organism in a large number of cases, but Horder's positive results (29 out of 32) are comparatively more numerous than those usually obtained. The micro-organism is generally isolated in acute cases, and less frequently in the chronic types, at any rate, until shortly before death. In our series positive results were obtained in 11 out of 27 cases; in Sir William Osler's, in 3 out of 6. The most common organism is a streptococcus of low virulence, which is closely "allied to the saprophytic streptococci of the alimentary tract" (Horder).

The characters of the vegetations are not specific, but in a general way the pneumococcic and gonococcic vegetations are numerous and small, while staphylococci produce a spreading ulceration.

ACUTE SIMPLE ENDOCARDITIS.

					Cases of Acute Rheumatism.	Cases of Endocarditis.
Sibson	..	..	..	..	325	161
Macrae	..	..	..	..	300	105
Church	..	..	..	..	889	494
Moore	..	..	..	..	100	99
Latham	..	..	..	..	136	74
					1750	933

First attacks (Church)—

Age	1 to 10,	cases	16,	endocarditis in	12,	75 per cent.
"	10 to 20,	"	109,	"	60,	55 "
"	20 to 30,	"	75,	"	38,	50 "
"	30 to 40,	"	36,	"	13,	36 "
"	Over 40,	"	8,	"	1,	12 "

Endocarditis was present in 149 of 150 fatal cases of acute rheumatism under the age of twelve (Poynton). In Macrae's series it was present in 45 per cent. of cases under the age of twenty, but only in 20 per cent. of cases over twenty years.

In Sibson's series murmurs made their appearance in 25 per cent. within the first week of the disease, and in 66 per cent. within the first fortnight.

In Macrae's series the mitral valve alone was affected in 77 per cent., the aortic valve alone in 5 per cent., and the two in 18 per cent. At St. Thomas's Hospital the corresponding figures were: mitral alone 85 per cent., aortic alone 3 per cent., and both in 12 per cent.

ACUTE MALIGNANT ENDOCARDITIS.

Age.					Horder.	Personal.	Total.
1 to 10	..	..	..	..	7	5	12
10 to 20	..	..	..	..	38	20	58
20 to 30	..	..	..	..	39	18	57
30 to 40	..	..	..	..	31	15	46
40 to 50	..	..	..	..	23	7	30
50 to 60	..	..	..	..	8	3	11
60 and over	..	..	..	..	4	2	6
					150	70	220
Sex :							
Male	..	..	..	..	79	46	125
Female	..	..	..	..	71	24	95

Site.	Osler. Cases 209.	Horder. Cases 118.	Personal. Cases 70.	Total.
Mitral valve ..	77	38	24	139
Aortic valve ..	53	22	19	94
Mitral and aortic	41	63	19	123
Tricuspid, etc...	19	14	7	40
Pulmonary, etc.	15	7	1	23
Mural	33	51	4	88
Right heart alone.. ..	9	—	—	9
Congenital de- fects	—	8	2	10

Previous Infections.	Horder. Cases 150.	Personal. Cases 57.
Acute rheumatism	} 72 {	24
Chorea		5
Tonsillitis		7
Pneumonia	1	10
Enteric fever	4	1
Diphtheria	—	1
Scarlatina	10	2
Influenza	2	2
Dysentery	1	—
Gonorrhea	7	—
Extreme oral sepsis	—	3
Atrophic rhinitis	—	1
Otorrhea	—	2
Lactation	—	2
Fistula	—	1
Fibroids (operation)	—	1
Ruptured bladder	—	1
Malaria	—	2

As the records are derived from hospital patients they are certainly defective in many respects.

Organisms isolated during Life.	Horder.	Personal.
Streptococcus	26	7
<i>B. influenzae</i>	5	—
Pneumococcus	5	1
Gonococcus	2	—
<i>Staphylococcus albus</i>	1	2
Doubtful variety	1	1

E. Libman has described a group of cases of subacute endocarditis from which he has isolated a streptococcus in 71 of 75 cases. He names the organism *Streptococcus mitis*. It is probably the same organism as the *S. viridans* and the *S. salivarius* of other writers.

In surgical cases the most common organisms are the *Staphylococcus aureus* and the *Streptococcus pyogenes*.

Horder, T. J., *Quart. Journ. Med.*, 1908-9, ii., 289. Osler, Sir W., *ibid.*, 219; Osler and McCrae's *System of Med.*, Lond., 1908, iv., 137. Libman, E., *Amer. Journ. Med. Sci.*, 1912, cxliv., 313; *ibid.*, 1913, cxlvi., 625; *Trans. XVIIth Internat. Med. Congress*, Lond., 1914, 195.

CHAPTER XIV

THE SYMPTOMS OF ACUTE ENDOCARDITIS

Simple Endocarditis

IN the majority of cases of simple acute endocarditis occurring in the course of rheumatic fever, the patient makes no complaint of any cardiac symptoms, and a valvular lesion is discovered only on routine examination of the heart. Occasionally palpitation or discomfort or even slight pain in the cardiac region may be experienced, but these are rare in the absence of pericarditis. There is seldom any dyspnoea, and the chief discomforts are the pain of the arthritis or the profuse sweating.

The Pulse.—The onset of endocarditis is, however, as a rule associated with definite evidence of cardiac embarrassment. The most constant signs are found in the *pulse*, which may be more frequent than the degree of fever would suggest, or infrequent, or irregular; and the abnormality may persist, even unchanged, though the fever and the arthritis subside, and convalescence is apparently attained. As an isolated symptom, an abnormal rate or rhythm is extremely suggestive, and may be present at a time when valvular murmurs are absent.

A girl of fifteen was admitted to hospital suffering from subacute rheumatism which had lasted for some six weeks. Within a week the arthritis had subsided, and the joints were mobile and painless. The heart seemed normal on examination, but the pulse rate continued to be high (90 to 100) after the fever had disappeared. A month later a very soft blowing murmur made its appearance for the first time at the

apex and the second pulmonic sound became accentuated. Four months afterwards the recognition of a typical pre-systolic murmur placed the diagnosis of organic valvular disease beyond doubt.

In many of these cases the recurrence of rheumatic symptoms (arthritis, tonsillitis, chorea, nodules) emphasizes the fact that the infection is not completely eradicated, but recurrence is extremely rare if the pulse rate has returned to normal figures. One fallacy, however, must be remembered. The pulse rate during the administration of salicylates is often within normal limits, but rises on their withdrawal, so that the pulse should be observed for at least a week after the administration of salicylates has ceased before any conclusion is reached (Fig. 178).

The pulse in endocarditis is occasionally *irregular*. This may be due to partial heart-block, which we have now

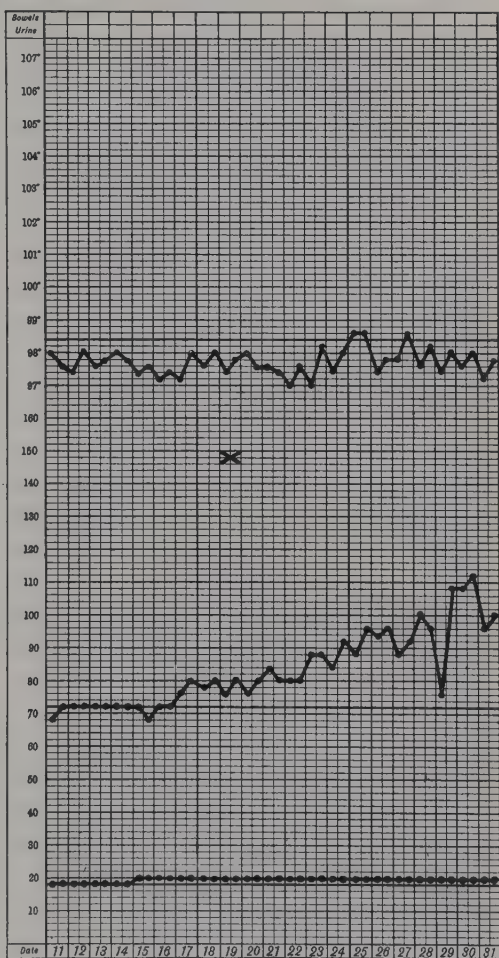


FIG. 178.—CHART SHOWING TEMPERATURE, PULSE RATE AND RESPIRATION OF J. MCK., AGED SEVEN. ACUTE RHEUMATISM; MITRAL ENDOCARDITIS.

Note rise of pulse rate on cessation of salicylate (X). The temperature had been normal for thirteen days.

observed in eleven cases. In one the defect persisted for nearly four weeks; in another it disappeared after six days, but recurred three days later, again lasting for six days; in a third it was present for several weeks before death; in the others it was transient, and only present for a few days. Heart-block in varying degree has been reported in gonococcic and streptococcic infections, and though it may occur in the infections apart from endocarditis (diphtheria),¹ the two are so commonly associated that their co-existence is extremely probable. An infrequent pulse is generally due to complete heart-block.

Extrasystoles may also occur. We have seen them in many patients, in three of whom nodal rhythm ultimately ensued. In another case a temporary partial block was succeeded a few days later by nodal extrasystoles. Extrasystoles are, of course, of common occurrence in various forms of cardiac disease, as well as in patients whose hearts are apparently normal, and their appearance during an attack of acute rheumatism can only be taken as suggestive of an acute cardiac lesion.

Auricular fibrillation occurs occasionally, but we have only seen it as a terminal event, and it obtains in so many dying hearts that its significance as a sign of acute endocarditis is slight.

The persistent form of nodal rhythm seems usually to be associated with acute endocarditis, which was found to be present in six of our cases on *post-mortem* examination. In another case of nodal rhythm the clinical course of the illness suggested a similar diagnosis, but no *post-mortem* examination was obtained. In one other case of nodal rhythm the lesions were perhaps chronic.

“**Cardiac Overaction**” is often present, the vehemence of the cardiac pulsations widely diffused over many interspaces being associated with a pulse which is small and soft to a degree which is quite out of proportion to the *apparent* strength of the heart. Cardiac overaction, however, owns several causes.

It is often present in hypertrophied hearts, in particular

¹ Fleming, G., and Kennedy, A. M., *Heart*, 1910, ii., 77.

those associated with aortic regurgitation. It is almost always present in nodal rhythm, and is frequently seen in cases of exophthalmic goitre when the pulse rate is frequent.

In nodal rhythm its origin is obvious. Auricle and ventricle are contracting simultaneously, and the auriculo-ventricular valves are consequently shut, so that the ventricular output must be limited; while the obstacle opposed to the auricles, and to the right ventricle in consequence, leads to vigorous though ineffective attempts to overcome the difficulty. A similar state of matters may also obtain in cases where the pulse rate is frequent, though the beats are of sinus origin; for if the rate of stimulus production is sufficiently rapid, auricular systole will occur before the *preceding* ventricular systole has ended, and a similar interference with the onward flow of blood will in consequence ensue (*see* Fig. 68).

The Auscultatory Evidence of endocarditis may be definite quite early in the illness, even before the arthritis has subsided or the associated fever gone. But a systolic murmur, it must be remembered, may be of functional origin, and is extremely likely to accompany the anæmia which is so rapidly produced by acute rheumatism. In some cases, too, the cardiac sounds are pure at first, and a murmur only develops during convalescence.

The *early auscultatory signs* may be very trivial, and a slight blurring or roughening of the first sound, or a short distant and muffled sound, or a short sharp, often loud, first sound, approximating in character to that of the normal second sound, may be the precursors of the permanent murmur of organic valvular disease. Murmurs are apt to be intermittent in the early stages, and may be distinct one day, and faint or gone upon the next, to recur in course of time. We have noticed this on many occasions, and in some cases which were examined *post-mortem* the valves were covered with very large vegetations, which by their bulk were quite capable of closing the aperture. In one case where an infective endocarditis involved a pulmonic valve which was congenitally defective, one of the longest and loudest diastolic murmurs which we have ever heard disappeared altogether for several days, though it returned before death.

The Occurrence of Pericarditis or the Appearance of Subcutaneous Nodules are additional evidence in favour of an endocarditis being acute. Pericarditis is almost invariably accompanied by endocarditis, though the degree of the two is by no means proportionate, and either may be severe when the other is slight.

Subcutaneous Nodules are generally considered to be indicative of gross cardiac inflammation, endocardial, pericardial or myocardial, an opinion with which our experience agrees, but the endocarditis is not necessarily severe. They are most common in early life. They are usually small (pin-head to a large pea), fairly firm in consistence, painless and not tender, and freely movable beneath the skin and upon the subjacent tissues. Tenderness may be present if they are mechanically irritated. They are most common about the back of the hand, the elbow, the knee, the scapula, and the occiput, occurring in relation to the fibrous planes, the ligaments, and the tendon sheaths; but we have seen them on almost every part of the body save the face. Comparable lesions as large as a cherry may occur in the muscles. In one case the patient frequently complained of pain in different muscles, which were tender to the touch. As a rule nothing abnormal could be felt, but sometimes an indefinite induration was apparent, which disappeared in a day or two. In another patient the lesions were very large, and one reached the size of a hen's egg, the surrounding tissues becoming brawny and infiltrated. The left sterno-mastoid was affected at one time, and the swelling which commenced at its upper extremity gradually spread downwards to the sternal insertion. Suppuration did not result, but the muscle remained indurated and hard for a week after the swelling had subsided.

In the majority of cases the nodules are discovered accidentally, and a routine examination then reveals several. In some patients fifty or more may be present at one time. They last for a variable period, a day or two, or several weeks, irrespective of their size, and resolution is complete. A migratory circinate erythema is a common accompaniment.

The Continuance of Fever after the arthritis has subsided, or a want of correspondence between its degree and that of

the joint affection, has as the most common cause an endocarditis. Endocarditis is generally present in cases where the arthritis relapses, even if the recrudescence is slight and temporary; and in cases where the other manifestations of the rheumatic poison, chorea, tonsillitis, erythema, etc., occur subsequently, perhaps in quick succession.

Malignant Endocarditis

The symptoms of the graver forms of the disease are very varied, as they may be referable to almost any organ. The endocarditis may be merely a part of a *general* pyæmia, a metastasis from the original septic focus, and may occasion little in the way of symptoms and have little direct connection with the fatal issue. A girl of nine, for instance, who had suffered from otitis media, noticed that her ears had again begun to discharge. A few days later a broncho-pneumonia developed, from which she seemed to be convalescing, when meningeal symptoms made their appearance and rapidly caused death. At the *post-mortem* examination vegetations were found on the aortic, mitral and pulmonary valves, and seemed the probable source of the meningeal infection.

In another group of cases endocarditis is merely a *terminal* event, occurring in a patient who is dying slowly from a chronic disease. In one of our patients who suffered from a pneumococcic endocarditis of the aortic and mitral valves and the left auricle, there was found *post-mortem* a quiescent tubercular lesion at an apex, a cancerous ulcer in the stomach, granular kidneys, and several strictures of the urethra! In another group *embolic* phenomena may be the first indication of the presence of serious cardiac mischief. A woman aged thirty-four, who had suffered from several attacks of acute rheumatism during adolescence, complained of epistaxis and of throbbing in the head during the later months of her fourth pregnancy. The confinement (triplets) in August was uneventful, and she made a good convalescence, though the throbbing continued. In the second week of October hemiplegia suddenly ensued, from which she died three days later. *Post-mortem* examination revealed an acute endocarditis of

the mitral valve and the left auricle, and a cerebral hæmorrhage which had ruptured into the ventricles.

The symptoms of the malignant cases are perhaps best illustrated by consideration of a number of cases in detail.

In some instances endocarditis is sub-acute but persistent, and serious symptoms only ensue after many months.

A well-grown girl of fifteen, who had had no serious illness, contracted an attack of acute rheumatism in December, 1910, which kept her in bed for two months. In April, 1911, chorea supervened and persisted for six weeks, and it recurred in June for nearly a month. In November she again suffered from rheumatism, this time for three weeks, and it returned in January, and in April, 1912, for a few days.

The cardiac phenomena which were recorded are instructive. On her first admission in April, 1911, the heart was of normal size and a mitral murmur was present. In June the murmur was longer and louder. In November the left heart was dilated for a time. In December a pulmonary infarct occurred, and she died of cardiac failure in February, 1913.

In these cases the arthritis may be trifling, even when the endocarditis is severe, and this is more likely to be the case in childhood than in adult life.

The early symptoms are sometimes extremely insidious. A postman, who had served throughout the recent war in France from the Retreat to the Armistice, was demobilized in March, 1919, and resumed his former occupation at once. In the spring of 1920 he began to feel weak, but he continued at work, and even played football until the middle of the summer, when he noticed that he became short of breath upon exertion. The weakness increased and he had to cease work in November. His only discomfort beyond weakness was a slight cough. In December his feet began to swell, and hæmorrhage occurred from the nose, lungs, bowel and kidney, and into the retina. The aortic and mitral valves were incompetent. The weakness rapidly increased and he died of cardiac failure in January. No cause for the infection could be detected.

Another patient, a clerk, aged twenty-six, was admitted into hospital in June complaining of weakness and breathlessness upon exertion of some months' duration. He had suffered from gonorrhœa and enteric

fever six years previously, and had never regained his former health. His feet were often swollen at night and he was unfit for exertion, but he was able to continue at work as a clerk. In February he caught cold and felt "run down." His work entailed a daily walk of four miles, and he felt exhausted at night. He became steadily weaker, but continued at his post until six weeks before admission, when he was forced to go to bed. The "cold" lasted from February to May. He now had frequent attacks of shivering at night, he felt fevered in bed, and often sweated profusely in the early morning. A fortnight before admission his right forearm and elbow became painful and tender, and this continued for a few days. Four days before admission the left hip and ankle became painful and this persisted.

On admission he was found to be a thin, pale, badly nourished man with flabby muscles and little subcutaneous fat. The left hip and ankle were painful and tender, and there was a small tender area of induration on the back of the left forearm. The right calf was painful, but seemed normal. There were several minute ecchymoses on the conjunctiva of the right lower eyelid, and a larger one on the left bulb, while ophthalmoscopic examination revealed a flame-shaped hæmorrhage in the left retina. The spleen was slightly enlarged, and the urine contained a small amount of albumen. The heart was but slightly enlarged, though there was a well-marked mitral systolic murmur. The pulse was regular, frequent, small and soft. He was slightly fevered at night. The leucocyte count never rose above 15,000.

His progress after admission was continuously in the wrong direction. Many embolisms occurred, chiefly in the limbs, but once at any rate in the spleen, and twice in the kidneys. He had several faint turns of a few minutes' duration, without obvious cause. The weakness increased, and in the middle of July œdema appeared in the legs. He sweated profusely at night, and had occasional rigors. A left hemiplegia occurred on August 25th, and he died on August 29th.

Post-mortem examination revealed an acute endocarditis of the mitral valve, two emboli in the right middle cerebral arteries, and infarcts in the kidney and spleen. From the latter the pneumococcus and *B. coli communis* were isolated. Blood-cultures during life had proved sterile.

In such a case, where the cardiac symptoms are slight, and the duration of the valvular lesion unknown, its relation to septicæmic symptoms may be difficult to establish. The occurrence of embolic phenomena, however, led to a correct diagnosis.

In some cases the symptoms point to the existence of gross lesions in the central nervous system, and a diagnosis may be impossible for a time. An old man was admitted into

hospital on account of cerebral symptoms which had developed somewhat rapidly, and as an aural discharge was found to exist the mastoid cells were exposed and drained, but without effect. *Post-mortem* examination showed that the symptoms were due to embolic occlusion of the cerebral vessels associated with malignant endocarditis of the mitral valve. The otitis media was quiescent. In the following case the correct diagnosis was considered probable on account of the sudden occurrence of cerebral symptoms associated with a cardiac lesion, fever and rigors. The significance of the occurrence of extrasystoles and nodal rhythm was not fully recognized until the *post-mortem* examination.

The patient was a man, aged twenty-four, whose previous health had been good, save for attacks of measles and enteric fever in childhood ; but he was often troubled with gumboils, and was frequently wet while at work. On July 1st he had a molar extracted on account of a gum-boil. In the evening he felt sick and shivery. These symptoms continued and on July 4th he had a rigor. On the 8th he went to the country, walking from the station to the house at which he stayed, a distance of two miles. He had a slight cough at this time with some discomfort in the left chest ; and on the 12th he had to go to bed as the pain in the side became worse. His left arm was stiff, and he was very breathless and at night had to sit up in bed. Next day he was better, and he was able to lie down in bed. On July 14th he felt better and went downstairs to breakfast without any difficulty. After breakfast he sat down on a chair, and on attempting to rise an hour later found that his legs were powerless and had no feeling in them.

On admission to hospital next day he was found to have little power in the legs, and to be quite unable to move the toes of the right foot. There was considerable anæsthesia in the legs. The power of the left arm was deficient. The right pupil was slightly smaller than the left. He was slightly fevered at night. He was quite clear mentally.

The tongue was dry and coated, and his mouth was very foul, the few remaining teeth being carious and septic. A mitral murmur was present, and the pulse was coupled from regularly recurring ventricular extrasystoles. There was a consolidation in the right chest, and friction at the left base. On the 16th he was better and the pulse was regular. On the 18th he was found to be semi-conscious with a full right hemiplegia. He died from cardiac failure upon the 27th. The pulse remained "single" until the 27th, when it was noticed to be again coupled for a short time. Tracings on the 23rd showed a nodal rhythm, with an *a-c* interval of 0.05 sec.

Post-mortem examination showed an acute ulcerative endocarditis of the mitral valve, engrafted upon a lesion of older standing, a some-

what chronic pneumonia of the right lung, infarcts in the spleen and kidneys and a softening in the left cerebral hemisphere.

The mitral valve was stenosed, the chordæ were short and thick, and the tips of the papillæ fibrous. The fibrosis spread upwards from the bases of the cusps towards the central fibrous body. The endocardium of the left auricle was thick and opaque, with a special area of thickening about as large as a shilling, on the contrafossal wall just above the valve.

Microscopic examination showed that the auriculo-ventricular node was the site of an acute inflammatory lesion which involved practically its whole extent. The bundle and the sinus node were normal. There was a well-marked myocarditis, involving both ventricles and auricles, but it was extreme only at the base of the mitral valve, and in the auricular tissues in the immediate neighbourhood of the auriculo-ventricular node.

The occurrence of ventricular extrasystoles and nodal rhythm was thus obviously the expression of a heightened irritability of the auriculo-ventricular node as a result of its implication in the myocarditis, leading to the establishment of this node as the dominant factor in maintaining the rhythm of the heart.

In chronic cardiac cases the onset of an acute process may be difficult to establish. A patient, aged twenty-nine, came under observation on account of fever accompanied by general symptoms which had developed six weeks previously. The fever was remittent, reaching 101–102° F. at night, and he had had occasional rigors and sweats. The organs other than the heart seemed normal. The heart was not enlarged, and a well-marked, somewhat musical ventriculo-systolic murmur was audible at the apex. The pulse was quiet and regular, running between 70 and 80. It was known that he had had mitral incompetence for many years since an attack of rheumatic fever in adolescence, but it had not troubled him in any way, and he had been on active service in Gallipoli and Palestine for two years without serious trouble. He had been on service at home for the preceding year. His medical attendant was well acquainted with his cardiac lesion, and was unable to detect any recent change in the condition. The infrequent pulse rate seemed to negative a cardiac cause for the fever. As time went on the pulse rate gradually rose, but never to any great height, and it was only shortly before his death some three months after the onset, that any appreciable alteration in the size of the heart and in the character of the murmur made the diagnosis clear. There were no embolic

phenomena at any time. Blood-culture showed the presence of small streptococci.

Considerable attention has recently been paid to the sub-acute forms of malignant endocarditis, which have been designated "subacute bacterial endocarditis." These cases are by no means uncommon, but though most commonly due to a non-hæmolytic streptococcus they may also follow an infection by *B. influenzae* and other organisms, and the condition cannot be considered a specific disease. A full account of the discussion on this subject at the British Medical Association will be found in the *B.M.J.*, 1920, ii., 301.

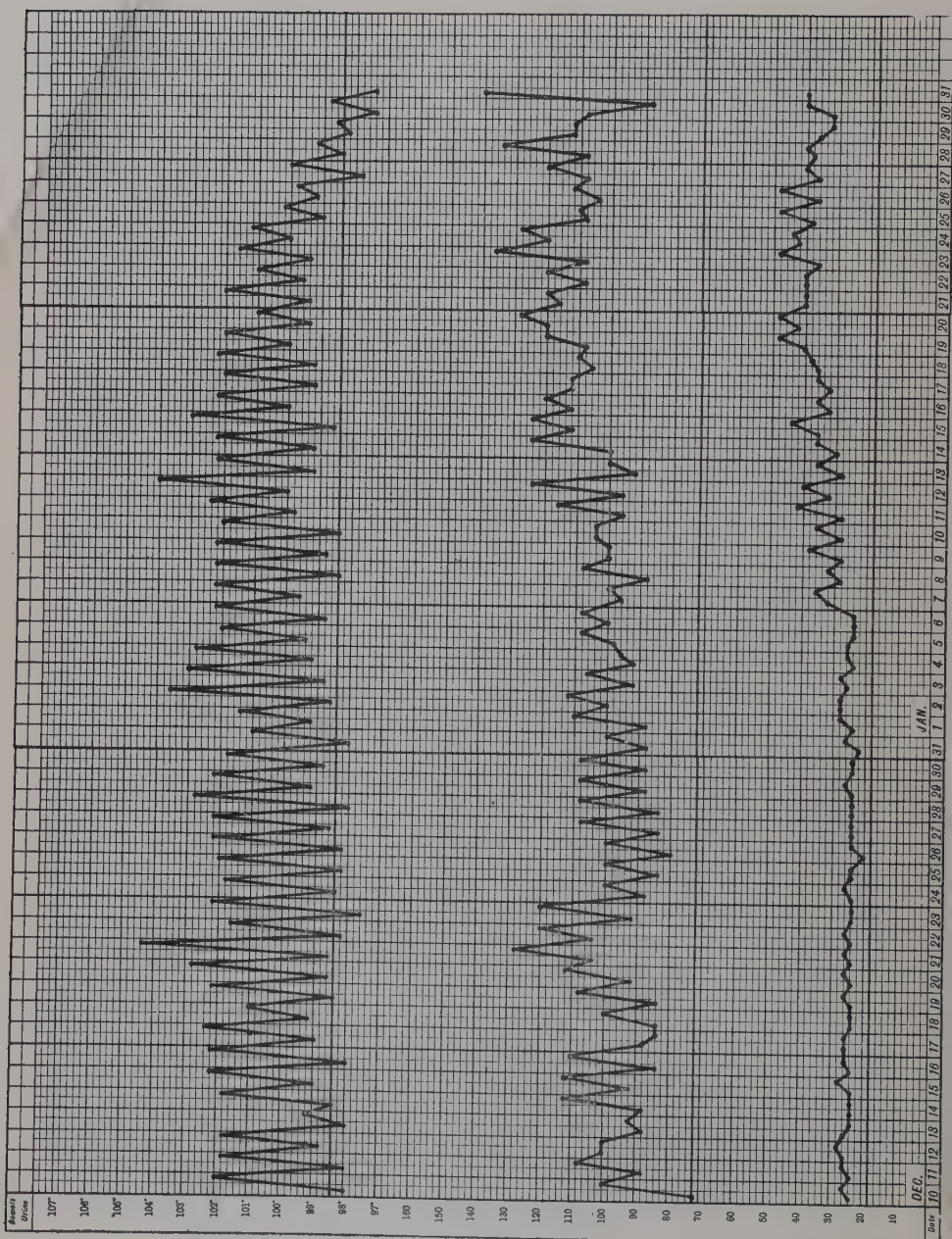
CHAPTER XV

THE DIAGNOSIS, PROGNOSIS AND TREATMENT OF ACUTE ENDOCARDITIS

The Diagnosis of Acute Endocarditis.—The diagnosis of acute endocarditis, though often self-evident, may be at times extremely difficult or even impossible, for in some cases the symptoms are general, and in some they point to the involvement of organs other than the heart. In our series the symptoms in 31 were cardiac, in 6 cerebral, in 6 pulmonary, and in 7 of a general kind; one patient died during an attack of chorea.

In the majority of cases *fever* is present (Fig. 179), but it is of no special type, and may be high or low, and continuous, remittent or intermittent; in the latter case rigors often occur. Fever may, too, be absent. This obtained in several of our cases, in one of which it was perhaps merely a measure of the debility of the patient; but in others the temperature was normal during the last few weeks of the illness. Fever is not infrequently absent during the acute stages of rheumatic endocarditis, and the temperature may even be sub-normal; sometimes the evening temperature rises occasionally to 99° or 100° F. without any obvious cause; or a sub-normal wave of “pyrexia,” lasting for a week or more, may be apparent on the chart (Fig. 180). Both these signs are significant.

The essential feature is the recognition of a valvular murmur, but the cardiac sounds may remain pure until some weeks after the onset of the illness (*see* p. 233). As a rule the auscultatory evidence of valvular disease is well marked, but previous observation may be lacking, or the presence of a pre-existing flaw may be known, and the separation of an acute from a chronic endocarditis is required. Horder has suggested that the occurrence of embolism and the isolation



of organisms from the blood are almost conclusive evidence of an infective endocarditis; but while this is probably accurate, it excludes practically all the cases of rheumatic origin, as well as many others in which blood-cultures are sterile, and thus restricts the recognition of the disease. It is on the cardiac evidence that most reliance must be placed.

We would again emphasize the importance of the *pulse rate* in the diagnosis. The pulse rate may be unduly frequent in fever, from emotion, from cardiac weakness, and in a few diseases, such as phthisis, exophthalmic goitre, rheumatoid arthritis, and diphtheria, apart from cardiac complications. But the absence of these factors can be readily determined by a few observations, and in their absence a frequent pulse rate is generally due to an acute cardiac lesion.

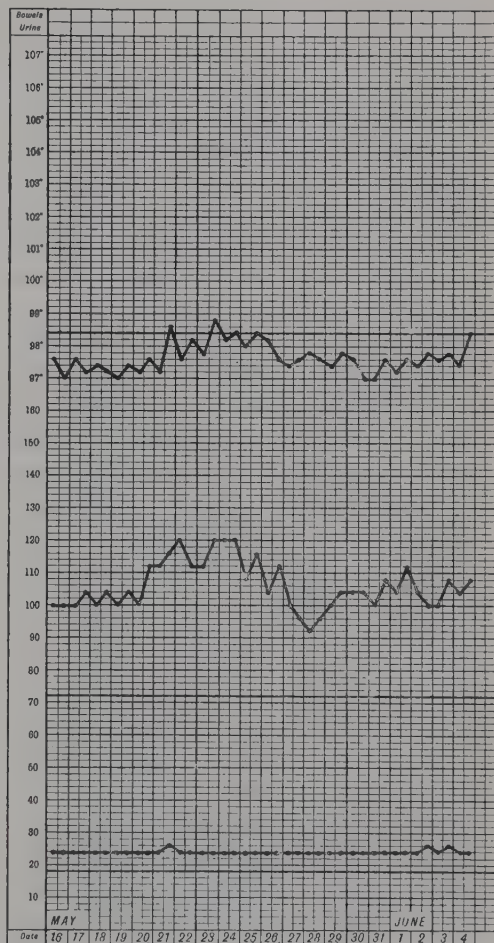


FIG. 180.—TEMPERATURE, PULSE AND RESPIRATION CHART OF H. O'K., AGED TWELVE: ACUTE RHEUMATISM; ACUTE ENDOCARDITIS.

Note subnormal wave of "pyrexia" and unduly frequent pulse rate.

The sudden appearance of *irregularity* has a similar significance. Heart-block is so generally due to a myocardial fault that its appearance, even in trivial and transient degree, suggests that an associated endocarditis is acute and not chronic; and the sudden appearance and continuance of extrasystoles during an acute illness, without obvious pulmonary complications or increase of cardiac weakness, point the same moral. Continuous nodal tachycardia seems to be associated most frequently with an acute endocarditis. Paroxysmal nodal tachycardia and auricular fibrillation have no particular significance with regard to the diagnosis.

The appearance of new murmurs is as a rule decisive, and the temporary disappearance of murmurs is equally suggestive, but one exception must be remembered. The characteristic crescendo murmur of mitral stenosis always disappears if auricular fibrillation or nodal rhythm occurs; and may disappear in partial heart-block.¹ The temporary disappearance of murmurs, while the sinus rhythm continues, is probably always due to an acute process.

Embolism is of frequent occurrence. It may of course occur in chronic endocarditis from the separation of clots in the auricles, but it is often multiple and recurrent in acute cases. In chronic valvular disease pulmonary embolism is more common; in acute endocarditis, embolism of the systemic arteries. The *spleen* is often involved (Horder, 47 cases) and becomes enlarged. Occasionally the enlargement is great even apart from embolism, and in two of our cases the lower border reached down to the umbilicus. The occurrence of a splenic infarct may be acutely painful, and friction may be audible from associated perisplenitis. *Renal infarct* is also common. Gross hæmaturia was present in 46 of Horder's cases and in 13 of our series; but, as Horder points out, the incidence is probably greater, for the lesser degrees are not always accompanied by *macroscopic* evidence of the presence of blood. Gaskell and Baehr have pointed out that this hæmaturia is often associated with a form of glomerular nephritis which is apparently characteristic of this disease. The *brain* is less frequently affected, but the cerebral vessels were occluded in 22 of Horder's cases and in 6 of our series.

¹ W. T. Ritchie, *Edin. Med. Journ.*, 1913, x., N.S., p. 410.

The result is most frequently softening, but hæmorrhage is not very rare. Meningitis occurred in 6 of our cases. In one of our patients the earliest symptoms were due to an embolus blocking the retinal artery.

Infective aneurysms may form. Horder reports 2 cases; in one the aneurysms were multiple, the right ulnar, the superior mesenteric, and the hepatic arteries being involved. In one of our cases, a boy of fourteen years, there was a small aneurysm of the aorta just above the valve, and a fusiform aneurysm, the size of a plum, at the lower end of the left iliac artery. Both the artery and vein were occluded, but the leg, though œdematous, showed no signs of gangrene. *Cutaneous manifestations* are not uncommon. Petechial eruptions are the most frequent, and were present in 43 of Horder's series and in 8 of our series. Subcutaneous nodules occurred in 4 cases in our series, and persistent erythema and urticaria in 3 instances. Fugitive tender indurations in the deeper structures have a similar significance.

Clubbed finger-tips are frequently present. They may, of course, occur in chronic pulmonary or cardiac disease, but their appearance during an *acute* illness is very suggestive of an acute endocarditis.

Gross visceral lesions are by no means uncommon, and the co-existence of pneumonia, pleurisy, pericarditis, etc., with a valvular murmur, should always suggest the possibility of an endocarditis being acute.

The occurrence of a leucocytosis has no particular diagnostic significance. It is always present in malignant cases save in the terminal stages, when it is sometimes absent; but it is merely evidence of a septicæmia, whose presence is clearly shown in other ways, and not of a cardiac infection. A count of over 25,000 is uncommon in the rheumatic forms.

The diseases with which acute endocarditis is most likely to be confused are *enteric fever*, *miliary tuberculosis*, and *septicaemia*. In each the general picture may resemble very closely those cases of endocarditis in which cardiac symptoms are absent. In all the onset may be insidious, the fever remittent, the spleen enlarged, and cardiac murmurs present. The occurrence of intestinal symptoms, an infrequent pulse rate, the rose rash, a leucopenia, suggest *enteric fever*. An

irregular fever, constipation, cyanosis out of proportion to the extent of any active or quiescent tubercular lesions in the chest, suggest *tuberculosis*. But every nicety of examination, cultures from the blood, urine and stools, and a wide view of the case may be necessary to establish a positive diagnosis.

The *septicaemias* sometimes simulate endocarditis very closely, for the latter is an arterial septicaemia. Deep-seated infections, sepsis in the urinary tract (particularly in children), and in the genital organs are those which are most difficult to establish. The occurrence of embolism in the systemic circulation, of cutaneous manifestations, and variations in the intrinsic action of the heart, may however lead to a correct diagnosis, though several weeks may elapse before they are manifest.

The Prognosis in Acute Endocarditis.—The prognosis in acute endocarditis is difficult to assess. The majority of rheumatic cases recover, but with damaged valves; while patients from whom positive blood-cultures have been obtained usually die.

In the individual case the risk must be assessed from consideration of the usual factors, the general condition of the patient, the height of the fever, and the rapidity of the pulse rate; and the prognosis should be guarded until the fever has gone and the pulse rate is within normal limits. The occurrence of rigors, embolism, and petechial eruptions; the appearance of oedema, or progressive dilatation of the heart; and alterations in the rhythm of the heart, are of serious significance. Complications, such as pericarditis, pleurisy and pneumonia add greatly to the danger. Delirium is generally the immediate precursor of death. But we have seen patients recover in whom each of these symptoms was present, and we are inclined to lay most stress upon the general nutrition of the patient. If this can be maintained the prognosis is never hopeless; but if it fails, the cardiac infection always progresses.

The Treatment of Acute Endocarditis.—There is no specific treatment of acute endocarditis, for, as has been shown, its causes are diverse. But the general indications

are clear, and the essential part of the treatment is **REST**.

It is, of course, impossible to secure physiological rest to the heart, for life ceases when the heart stops beating; but absolute confinement to bed in the recumbent posture diminishes the frequency of the cardiac contractions and ensures a minimal blood-pressure; for both the pulse rate and the blood-pressure are appreciably raised even by sitting up in bed. Absolute rest necessitates full nursing, the patient doing nothing that can be done for him by a nurse, and being fed and washed and dressed with as little voluntary movement as is possible. The use of a bed-pan and urine bottle is essential. The rest, too, must be long continued, for even after all the symptoms have disappeared and the pulse and temperature curves are normal, the valves are infiltrated and soft, and resolution is incomplete; and the size of the ultimate scar depends upon the degree of movement of the cusps.

In too many instances the duration of the rest allowed is insufficient. It is impossible to assess accurately the extent of the inflammation, but analogy from lesions elsewhere, such as pneumonia and fractures, shows that the rate of resolution varies considerably in individual cases; and the only wise plan is to play for safety and rather to prolong the rest unnecessarily than allow exercise at a time when it will, or may, cause harm. The effects of exertion on the incidence of endocarditis in acute rheumatism are well known, and the lesions are less common and less severe in cases where the severity of the arthritis has driven the patient at once to bed, as contrasted with the milder cases where the patient has been able to walk about for days or even weeks.

Absolute rest, then, is indicated until all symptoms have disappeared, and the rate and rhythm of the pulse are normal. When this has occurred, the patient may be allowed to sit up in bed for short periods, the size of the area of cardiac dullness and the pulse rate being carefully watched; and the periods may be increased gradually if no untoward result follows. He should, however, be confined to bed for at least two months longer, and for another month merely moved on to a couch, before he is allowed to stand up and walk. Restrictions as to exercise must be continued for at least three to six

months longer, and violent exercise, such as games, interdicted for a year or so afterwards.

Massage is useful during the period of rest, and may be commenced as soon as the patient is allowed to sit up, the massage being at first slight and of short duration, and only increased very gradually. Every effort, too, should be made to improve the general health, and plenty of fresh air and a variety of food utilized with this in view. In suitable weather much time may be spent out of doors.

The results obtained by these means are surprising. One patient, aged forty when first seen, had a heart so full of murmurs that no valve could be pronounced sound, but its size was normal. He was a lawyer in busy practice who took much exercise, and he had never had any cardiac symptoms since his attack of acute rheumatism twenty years before. But at that time he was for six months in bed and for six months on a couch, and he was not allowed to walk quickly for another year.

From the clinical standpoint cases of endocarditis may be divided into three groups : (1) Those which are of rheumatic origin ; (2) those in which blood-cultures reveal the presence of bacteria ; and (3) those in which blood-cultures are sterile, though the disease seems to have no connection with acute rheumatism.

1. The incidence of endocarditis in acute rheumatism, as gauged by statistics in the literature, does not seem to be appreciably lessening, so that it appears that salicylate treatment has little effect in preventing this complication. Every one knows how difficult it is in some cases to overcome the tendency of the arthritis to relapse in this disease ; and most of us have watched cases of rheumatic endocarditis progress steadily, notwithstanding salicylate treatment. It has, in consequence, been suggested that the failure of the salicylates indicates a non-rheumatic infection. The inference, however, does not seem justifiable, for cases of malaria or syphilis may run a malignant course in spite of treatment by quinine or mercury ; and the obvious conclusion is that acute rheumatism may run a malignant course. A progressive lesion merely indicates a severe infection, not necessarily one different in nature.

The action of large doses of salicylates upon the heart, slowing its rate and weakening its action and lowering the blood-pressure, has been held to contra-indicate their administration in cases where the heart is involved. If there is any evidence of cardiac weakness the contention is obviously sound, but in its absence the drug should be exhibited, as, apart from the question of a specific action upon the infection, the lessening in pulse rate and the lowering of blood-pressure are beneficial to the local lesion. Large doses are, however, unwise, but 60 to 90 grains may be given daily to an adult in combination with equal doses of sodium bicarbonate.¹

Salicylate poisoning is comparatively infrequent. The lesser degrees are most frequently observed in patients who are suffering from high blood-pressure and defective elimination. In any case attention should be paid to the smallest manifestations of intolerance, and the drug should be intermitted if they appear.

2. The isolation of bacteria from the blood necessarily raises the question of specific treatment, and it seems reasonable to administer the corresponding antiserum in these cases, even though one must as a rule employ a polyvalent serum, and not one specifically related to the particular organism. The results which have been obtained, however, are extremely disappointing, and only one or two cases have been recorded in which recovery followed. Our own experience has been uniformly unsatisfactory.

Vaccine treatment, too, has had equally poor results, and it seems extremely doubtful if good results will ever be obtained in this way. The production of antibodies, which is the aim of this treatment, takes place in disease in response to the presence of bacteria or of their products, and may fail to be sufficient if the infection is local and absorption minimal; or if the infection is intense and the defensive processes are overpowered. Of all the conceivable sites of infection, the endocardium is surely that most certain to produce a general intoxication, and we fail to comprehend how in these general

¹ The administration of alkali in an amount sufficient to render the urine alkaline, and the prevention of constipation, obviate the risk of salicylate poisoning (Lees), which is perhaps due to an acid intoxication.

“arterial” septicæmias an additional dose of the poison can be of benefit to the patient.

In four cases, however, recovery has ensued after treatment of this kind, and although the satisfactory result may not have been due to the specific treatment, the uniformly fatal termination in cases in which such treatment has not been tried should perhaps encourage further experiments.¹

3. In the third group the indications for treatment of a specific kind are lacking. The salicylates have generally been already tried and have failed, and serum or vaccine treatment are negatived by the failure to isolate a bacterium. General treatment of the kind that has been already laid down, complete rest, fresh air, an abundant dietary (solid food can often be well digested by fevered patients), is of course already in action.

It seems probable that in many of these cases continual reinfections of the blood-stream (and of the valves) are occurring from local septic foci which may exist in different situations. The tonsils are often large and deeply pitted; the nose or middle ears, the antrum or the frontal sinuses are septic; or the teeth are carious and the mouth is foul. The urethra, the bladder, the uterus, etc., may be infected. The influence of such septic foci in the production of a visceral infection is difficult to estimate, but an impression is gradually arising that they are of greater importance than has hitherto been recognized; and that little good result is likely to occur unless the reinfection of the valves is stopped by eradication of the source of the reinfection. The general condition of the patient, or the nature of the primary focus, may make it difficult to secure asepsis, but we have on two occasions seen permanent improvement ensue in cases which seemed to be progressing downhill, after removal of enlarged tonsils; and on one occasion a great, though unfortunately only temporary, relief after the removal of some dozen carious teeth. From the preventive point of view, in any case, such measures are

¹ Sir James Barr, W. Blair Bell, and Captain S. R. Douglas, *Lancet*, Lond., 1907, i., 499. A. Latham and E. L. Hunt, *Proc. Royal Soc. Med.*, Lond., 1910, iv. (Clinical Section), 14. G. Joehmann, *Berlin klin. Woch.*, 1912, 436. H. Hemstead and T. J. Horder, *Lancet*, Lond., 1913, i., 10.

urgently required, and no "rheumatic" patient should be allowed to harbour septic foci, even in the absence of cardiac disease.

Many drugs have been advocated in these cases—horse serum (to supply complement), arsenic, mercury, quinine, silver in the colloidal form, etc. We have once or twice thought that collargol injected intravenously, and applied as Crede's ointment to the skin of the trunk, was responsible for improvement. In most cases it has no effect. In two instances the intravenous administration of another form of colloidal silver was undoubtedly harmful, and hastened the fatal issue. The most important factor is probably the digestion; and so long as the nutrition of the patient can be maintained or improved, recovery may be hoped for; with anorexia or digestive disturbance the progress is invariably in the wrong direction. We have seen recovery ensue in a case where the fever persisted for 74 days. The patient was eventually, eight years later, killed in action, after two years' service in France. During his illness his digestion was always satisfactory.

The occurrence of symptoms of cardiac weakness is always of grave significance. They must be combated by the usual means.

CHAPTER XVI

CHRONIC VALVULAR DISEASE

The Lesions.—Chronic valvular disease of the heart interferes in greater or less degree with the proper working of the valves, and thus imposes an extra strain upon the heart ; but as the lesions differ in their grade and are not necessarily confined to the cusps but may involve the aorta, the pulmonary artery or the muscle as well, the ultimate result varies in different cases, and the interference with the cardiac action may be considerable or of little moment.

The conditions which are found *post-mortem* are of various kinds. The normal, soft, pliable, translucent valve is altered, and the cusps are hardened, stiffened and opaque, the change being in some cases uniformly distributed throughout, and in others confined to, or most extreme at, the free margins or the lines of attachment. Calcareous (Figs. 181, 182) and atheromatous deposits are not uncommon, and although usually merely an addition to a general change, are sometimes an isolated lesion in a fairly normal cusp. The calcareous



FIG. 181.—CALCAREOUS INFILTRATION OF AORTIC VALVE (GLASGOW ROYAL INFIRMARY MUSEUM).

deposits are occasionally so large that by their mere bulk they interfere with the proper action of the valve, and may prevent its closure or obstruct the valvular orifice.

The cusps may be shortened or adherent to each other ; and while shortening produces incompetence, adhesion causes narrowing of the orifice ; the two results frequently co-exist.

The ultimate result varies. Sometimes the cusps are more or less uniformly joined together and a funnel or dome-shaped appearance is produced; sometimes the longitudinal shrinkage is extreme, and a diaphragm results (Fig. 183). The chordæ tendineæ, and even the muscoli papillares, may be implicated in the fibrosis of the mitral valve (Fig. 188), and the whole apparatus may be welded together into a single mass (Figs. 184, 187). The orifice is often greatly narrowed, and may be rounded, or linear, or stellate.

In many cases warty outgrowths of varying size and consistence are visible at the margins of the cusps; in others erosion and loss of substance are the main feature and the edges are puckered and uneven. The line of attachment may be eroded and part of a cusp may be freely mobile in the blood-stream; or, if calcified, project stiffly into the lumen. These results are most common in the aortic valve, for destruction of the chordæ ten-

dineæ of the mitral valve usually produces so great an interference with the cardiac action that death ensues during the acute stage.

The cusps may be perforated through their substance. This is most commonly the result of acute endocarditis, but sometimes follows severe muscular effort or an external trauma. Those pathological perforations must not be confounded with fenestration of the cusps, a congenital defect of no importance, which does not interfere with the action of the valves. The distinction is easy, as the fenestræ are situated close to the margins of the cusps, and on surfaces which are opposed during closure; while the edges are unaltered, and neither

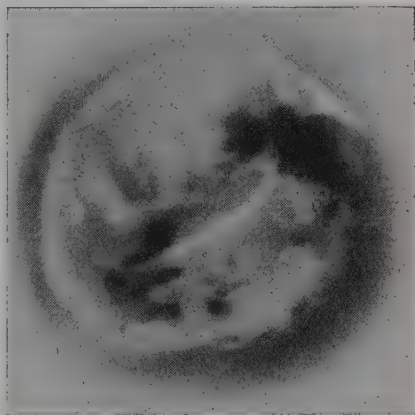


FIG. 182.—RADIOGRAM SHOWING CALCAREOUS DEPOSITS IN THE MITRAL VALVE. K.M.C. (GLASGOW ROYAL INFIRMARY MUSEUM).

thick nor hard. In the former the cusps are abnormal, and the perforations are at the centre of the cusps or near their attachments.

In the majority of chronic aortic defects the edges of the cusps are more or less inverted towards the sinus; in rare instances the

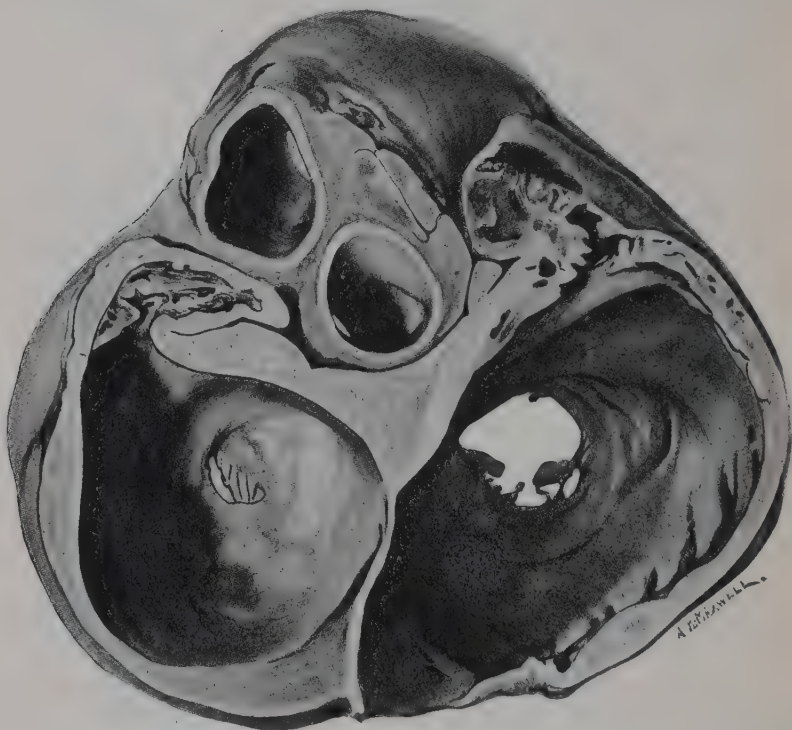


FIG. 183.—TRANSVERSE SECTION OF HEART, VIEWED FROM ABOVE (WESTERN INFIRMARY MUSEUM).

The mitral valve is greatly, and the tricuspid valve slightly, stenosed. Both auricles are greatly dilated.

edges are everted.¹ In the latter case (Fig. 185) the cusps are usually voluminous rather than shortened, but, though thickened, are so soft that they fall downwards towards the ventricle and impose no obstacle to the regurgitation of blood. Rarely there is necrosis or ulceration, and vegetations, if present,

¹ Cowan, John, *Glasgow Med. Journ.*, 1902, lviii., 198.

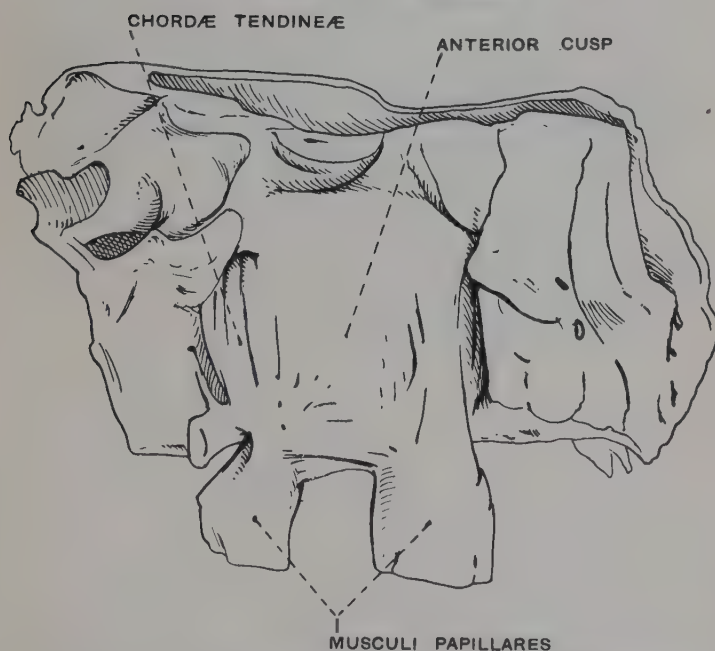
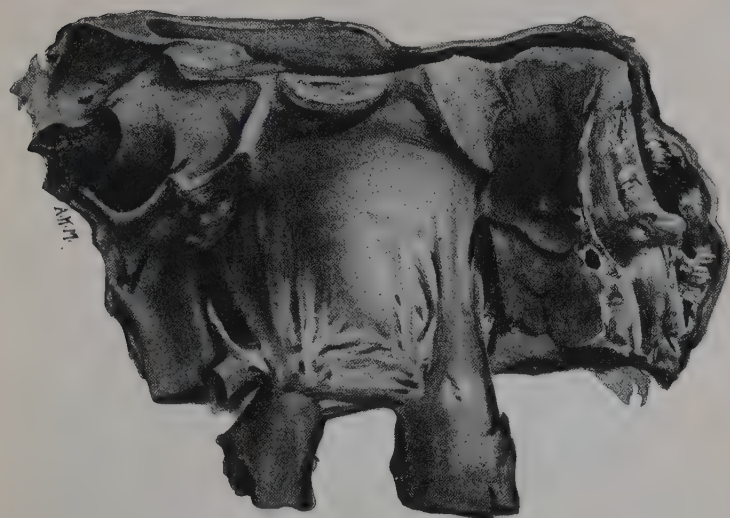


FIG. 184.—FUNNEL-SHAPED MITRAL ENDOCARDITIS (WESTERN INFIRMARY MUSEUM).

The mitral cusps, the chordæ tendineæ, and the muscoli papillares are all welded together,

are generally scanty and small. Microscopic sections show that the connective tissue of the cusps is loose and degenerate, with patches of leucocytic infiltration which are most numerous at the base of the valve and in the aortic wall; the vessels here show inflammatory changes and a narrowed lumen, interfering with the nutrition of the valve and producing degeneration of the new-formed connective tissue.

The lesions frequently extend beyond the valve. In chronic disease of the aortic valve, the aorta is often affected, showing either a diffuse thickening or a patchy change; the aortic orifice may be so greatly dilated that even cusps of normal size would be insufficient to occlude it. The mitral orifice, on the other hand, is more often narrowed than dilated, for

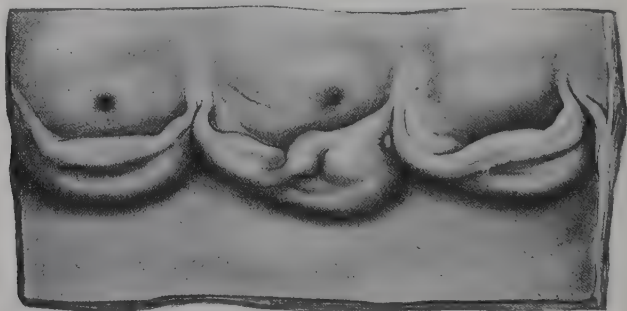


FIG. 185.—FLABBY "WASH-LEATHER" THICKENING OF AORTIC VALVE, WHICH WAS NOTABLY INCOMPETENT (WESTERN INFIRMARY MUSEUM).

the inflammatory spread into the muscle in the course of cicatrization leads to narrowing. The auricular wall may be affected (Fig. 186). Valvular defects are rarely pure. Stenosis is almost always associated with some degree of incompetence; and conversely incompetence is generally accompanied by some degree of narrowing. In aortic cases incompetence is the rule, and stenosis is minimal and rarely extreme. In mitral cases stenosis is the rule, and incompetence co-exists; for the anatomical division of the mitral valve into an anterior and a posterior cusp is inaccurate, as it is really a circular curtain with special developments of certain parts; and if it is uniformly involved, the lumen is necessarily narrowed by the contraction of the scar tissue.

The close proximity of the valves to particular structures is an important factor in the ultimate progress of the case. The orifices of the coronary arteries are frequently involved in aortic disease, and the auriculo-ventricular node and bundle are often damaged in mitral and in aortic lesions. The interference with the nutrition of the heart produced by the former, and the disturbance of the cardiac rhythm entailed by the

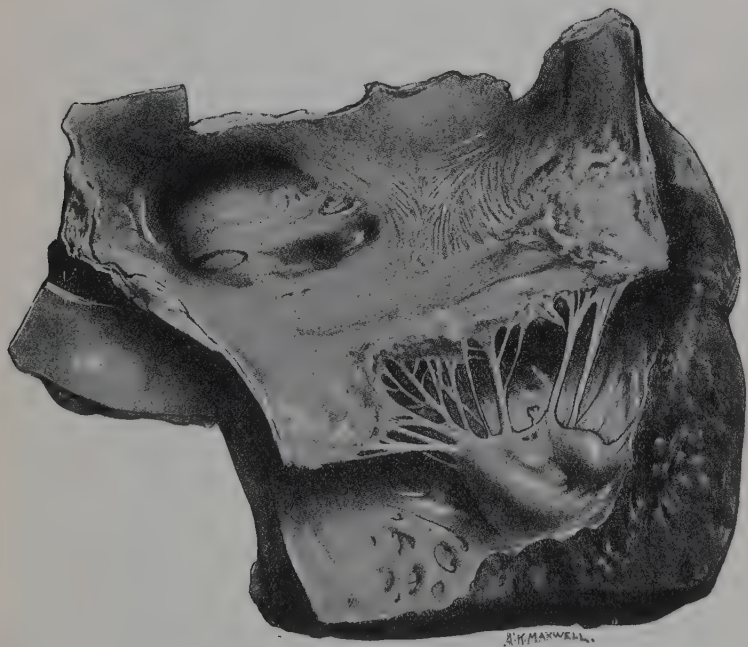


FIG. 186.—MITRAL ENDOCARDITIS, SHOWING A LARGE HEALED SCAR ON THE WALL OF THE LEFT AURICLE (*cf.* FIG. 177) (GLASGOW ROYAL INFIRMARY MUSEUM).

latter, imprint special characters upon the symptoms of these diseases.

The Etiology of Chronic Valvular Disease.—There is considerable difficulty in determining the precise causes of chronic valvular lesions. More than one cause is frequently active; the effects of different causes are very similar; the chronicity of the disease prevents accurate observation of its progress; and the ultimate result depends on the site

of the affection rather than on the nature of the cause.

Chronic valvular disease may follow (1) acute endocarditis ; (2) syphilis ; or (3) mechanical strain ; or may be (4) merely a part of a general cardio-vascular degeneration, or (5) a defect of development.



FIG. 187.—MITRAL STENOSIS.

Woman aged thirty-nine, who developed Nodal Extrasystoles and subsequently Auricular Fibrillation.

The cusps of the mitral valve and the chordæ tendineæ are thick and welded together. The left auricle is dilated ; the auricular appendix is filled by a thrombus.

1. *Acute Endocarditis*.—*A priori* it seems probable that a slight acute endocarditis may resolve completely, but pathological data are necessarily lacking, and the clinical evidence is insufficient to prove the point. In the majority of cases

the lesion heals in course of time, but resolution is imperfect and a scar is left behind, which, from the constant movements of the parts, is larger than it would have been if it had been situated on a less mobile site. Necrotic lesions of course cannot be replaced, so that in practically every case of acute endocarditis a valvular defect is left. If the lesion is small,



FIG. 188.—MITRAL VALVULAR DISEASE.

The left auricle and ventricle are laid open to show the dilatation and hypertrophy of the auricle, the thick, shrunken mitral valve and chordæ tendineæ, and the hypertrophy of the ventricle. From a woman, aged sixty-three, who presented complete auriculo-ventricular block.

the mechanical defect may be minimal, but the damage makes the valve more vulnerable, and more liable to be seriously injured by causes which would produce little effect upon a healthy valve. The free margins of the cusps are most affected, and the lesions may be confined to these parts, or may involve the whole of the cusp.

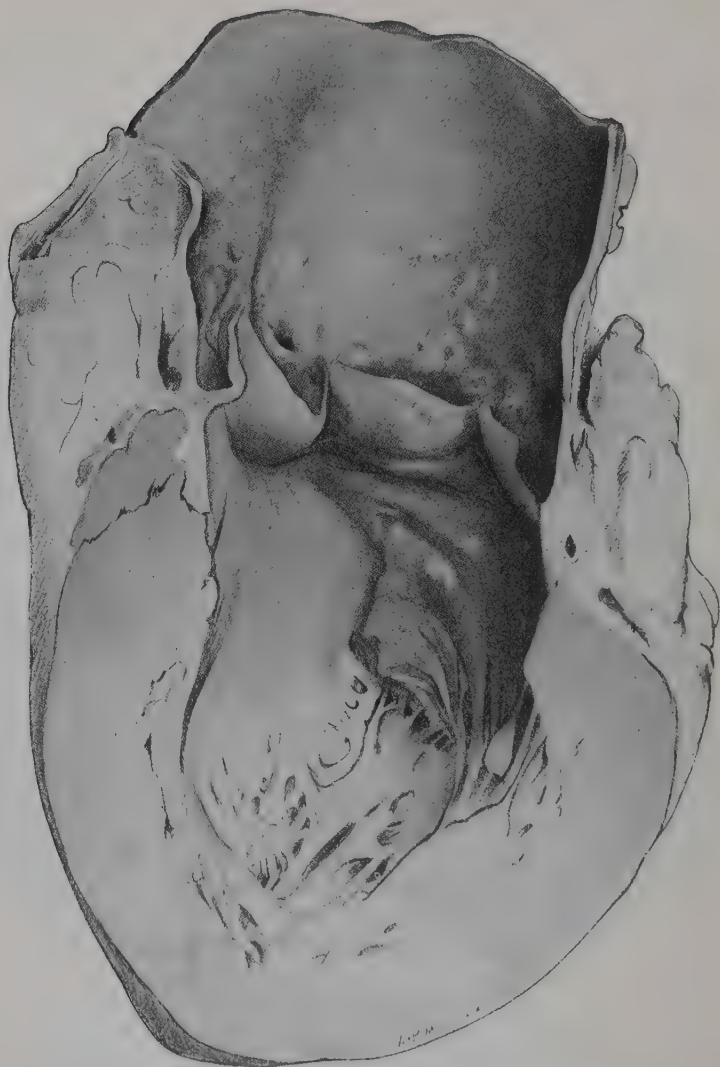


FIG. 189.—SYPHILITIC AORTITIS, WITH INVOLVEMENT OF AORTIC AND MITRAL VALVES (GLASGOW ROYAL INFIRMARY MUSEUM).

2. *Syphilis*.—The effects of syphilis are only gradually being appreciated at their true value. The specific arterial lesions have but recently been distinguished; a syphilitic scar upon a valve closely resembles those produced by other

causes; and the chronic disease can only be recognized by the presence of other syphilitic stigmata, though the acute lesions are distinctive. These are well shown in Fig. 189. The patches on the aorta are characteristic (Figs. 29, 271); and the thick yellow nodules upon the aortic and mitral cusps are identical in appearance. The damage is more widespread than would appear from mere inspection of the surface; and the cut surface of the cusp to the left of the drawing (Fig. 189) shows a widespread thickening. Syphilitic nodules tend to break down in their centre, and the resultant scar leads to shortening and puckering of the cusps. The valvular disease may occur at any stage of the disease, and though most common as a late sequel, may also obtain comparatively early if treatment is defective. Accurate data are difficult to procure, but in one case where the patient died with aortic and mitral disease, the symptoms of cardiac distress appeared two years after the chancre, and two years before death. No specific treatment had been undergone. The patient was inclined to athletic exercises, and the symptoms may have been accelerated by physical exertion. (*See also p. 270.*)

3. *Mechanical Strain.*—The relations of mechanical injury to chronic valvular disease are equally difficult to assess, though isolated cases have proved beyond doubt that acute lesions may be produced in this way. Sir Clifford Allbutt¹ quotes many examples. A healthy man, aged thirty-six, was crushed between a cart-wheel and a post, and died a few days afterwards; the healthy aortic valves had been torn away from the healthy aorta. A man, aged nineteen, had an aortic cusp ruptured from the free margin to the base in consequence of a blow upon the chest. A man, aged twenty, fell from a scaffold and developed well-marked aortic regurgitation. And in a certain number of cases the severe strain of some physical exertion has been so rapidly followed by serious cardiac symptoms that the association seems clear. A porter, for example, who was lifting a heavy trunk on to a barrow, suddenly became exceedingly breathless, and when seen a few days later was the subject of well-marked aortic regurgitation. A miner, aged thirty-two, when lifting a heavy stone was suddenly seized with severe pain in the chest,

¹ Allbutt and Rolleston's *System of Medicine*, Lond., 1909, vi., 425.

and subsequently found to have well-marked aortic regurgitation. No other cause than the strain could be discovered. But such cases are rare, and in most the evidence points to valvular disease being antecedent to the trauma, which produced gross results in consequence of some local weakness.

Two other groups of cases of mechanical strain require consideration. In some occupations (moulders, draymen, riveters, *et alii*) severe physical exertion is constantly undertaken, with, in consequence, high blood-pressure during the period of stress; and in some diseases (arterio-sclerosis, chronic renal disease) the blood-pressure is constantly above normal. The question of the influence of an intermittent or a constant high blood-pressure upon the valves naturally arises.

There is no doubt that men in arduous employments grow old before their time; the Clyde shipbuilders are rarely in active work after the age of fifty-five; and the main damage falls upon the heart and the arteries. But the habits of these men are often loose. Labour involving sweating is often associated with the consumption of large quantities of alcohol; and syphilis and other infections and intoxications not infrequently co-exist; so that it is difficult to ascertain the exact value of the mechanical factor.

The influence of intermittent strain can hardly be determined by pathological examination, though microscopic hæmorrhages and local scars have been found in such cases. A correct appreciation of the frequency of its action can only be gauged by clinical data on the large scale. But there is already much information available with regard to the occurrence of chronic valvular disease in cases of high blood-pressure, and it has been found that the valves are very frequently damaged.

The connection between *mitral* and renal disease was first noticed by Barclay in 1848, and several writers have in the interval investigated the question from *post-mortem* data. Goodhart found that, of 97 cases of renal cirrhosis, 47 had mitral lesions; while of 215 cases of (clinically) primary mitral disease, 31 had cirrhotic kidneys. Newton Pitt found that, of 542 cases of renal cirrhosis, 33 had mitral stenosis, and that 33 per cent. of 115 cases of mitral stenosis had renal

cirrhosis. In a series of 335 cases of renal fibrosis (capsules adherent; surface more or less granular), reported by Fleming and one of us,¹ 60 (17·9 per cent.) had organic disease of the mitral valve, and of 93 cases of mitral disease 60 (64·5 per cent.) had fibroid kidneys. It seems certain, too, that the association is not accidental, for in a corresponding series of 807 cases, in which the kidneys were not fibroid, only 33 (4 per cent.) had organic mitral disease.

The relationship between mitral and renal disease is not always the same.

(a) In some cases it seems clear that the cardiac lesion is primary. A man who died at the age of twenty-one had suffered from chorea at the age of three; the mitral valve was notably hard and deformed, while the kidneys were only slightly affected. A woman who died at the age of forty-eight had suffered from acute rheumatism at twenty-two; the mitral lesion was extensive and the interstitial nephritis "early." While a woman who died at the age of thirty-five had had several attacks of acute rheumatism before twenty-eight; and her kidneys, though granular, were still large. In all three cases the symptoms were cardiac.

It seems improbable that the renal lesion is due to chronic venous congestion as has been suggested, for if that were the case the liver should suffer as frequently as the kidneys; but in our series of 93 cases of organic mitral disease, the kidneys were affected in 60 and the liver in but 9. The causes of renal cirrhosis are still unknown, but there is considerable evidence in favour of the view that it may be due to faulty intermediate metabolism and the consequent irritation of the kidneys by abnormal substances which are excreted in the urine; and chronic venous congestion of the digestive organs is extremely likely to derange their proper action, and is not infrequently accompanied by gastro-intestinal catarrh, which will exert its own special action.

(b) In another group of chronic mitral cases the usual causes of acute endocarditis are conspicuous by their absence. A woman who died of mitral disease at the age of forty-five would not admit having had any illness whatever before the onset of that which ultimately proved fatal. A woman,

¹ *Quart. Journ. of Med.*, 1912, v., 309.

aged forty-three, had had measles and scarlatina in childhood, but had always been healthy until a few winters before her death, when she became liable to bronchitis and short of breath on exertion. A man, aged forty-seven, had had no previous illness, with the exception of an attack of "gastric" fever at the age of fourteen. And there are a small number of cases on record in which mitral stenosis has been observed to develop in the absence of all acute disease (Sansom). It is generally agreed that the most frequent causes of those chronic cases are arterio-sclerosis and chronic nephritis, and in these three cases the kidneys were more or less granular and their capsules were adherent.

Huchard has outlined the process by which the lesion is produced. The vessels of the valve are frequently degenerate, their lumen is diminished, and minute hæmorrhages are not uncommon. As a result the overlying endothelium is lost and fibrin is deposited upon the uncovered surface; and the constant movements of the part produce a "callus" which is large and out of proportion to the original lesion. Kennedy, too, has investigated the point and finds that the basal portions of the mitral cusps are almost always thickened and fibroid in chronic renal disease (Figs. 190, 191, 192).

It thus seems evident that in some cases the renal lesion is the cause of the mitral one, but it is improbable that this is the common sequence. It is true that the incidence of mitral disease in cases of renal fibrosis is much greater than in cases where the kidneys are normal, but the incidence is much greater in women than in men,¹ a fact which suggests that the connection often arises in other ways.

(c) Mitral and renal disease may conceivably be due to the same cause, and though actual proof of the association is lacking, the evidence in its favour is suggestive. Newton Pitt and Sansom maintained that acute endocarditis and

¹TABLE SHOWING THE INCIDENCE OF MITRAL DISEASE IN CASES OF RENAL FIBROSIS.

	Renal Fibrosis.	Mitral Disease.	Per cent.
Men	201	27	13·4
Women	134	33	24·6

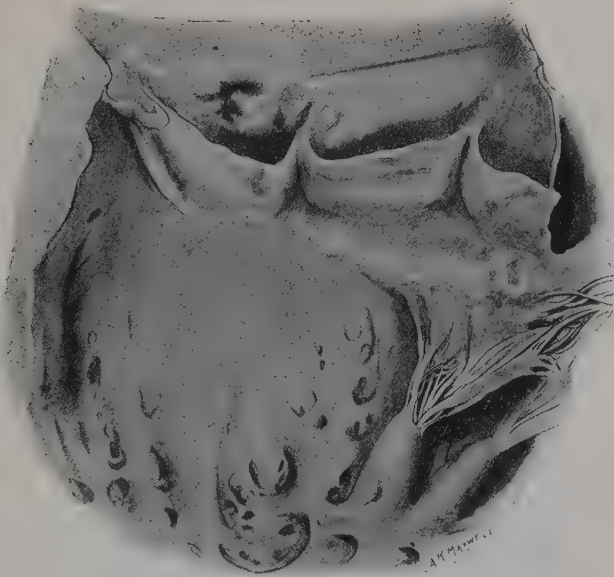


FIG. 190.—CHRONIC RENAL DISEASE: PATCHY THICKENING TOWARDS THE BASE OF THE MITRAL VALVE (GLASGOW ROYAL INFIRMARY MUSEUM).



FIG. 191.—CHRONIC RENAL DISEASE: DIFFUSE THICKENING OF THE BASE OF THE MITRAL VALVE (GLASGOW ROYAL INFIRMARY MUSEUM).

nephritis might both originate during the puerperium, and so initiate the chronic disease, and Rolleston has suggested that they might result from syphilis. Acute syphilis at the present day rarely causes death, and its real importance as a cause of chronic mischief is apt to be obscured by its chronicity, unless we remember the possibility of its presence in cases where the etiological factor is in doubt, and employ the Wassermann test to affirm or deny its presence. We have



FIG. 192.—CHRONIC RENAL DISEASE: DIFFUSE THICKENING OF THE BASE OF THE MITRAL VALVE, WITH SUPERADDED ACUTE ENDOCARDITIS OF THE AORTIC VALVE (GLASGOW ROYAL INFIRMARY MUSEUM).

observed one acute case in which both the kidneys and the cardiac valves were affected. The patient had contracted a chancre four years before, for which he had not received specific treatment. The arteries were extensively diseased, the aortic valve was notably deformed and grossly incompetent, the mitral valve was dilated and the cusps were shortened and thickened, and the cortex of the kidneys was slightly

atrophied and their surface somewhat granular. Scarlatina, too, is a not infrequent cause of acute endocarditis and acute nephritis, and might affect the heart and the kidneys simultaneously.

The work of Gaskell and Baehr has thrown a new light upon the matter. The subacute forms of acute endocarditis from which Horder and Libman have so frequently isolated streptococci are very often accompanied by glomerular changes in the kidneys. An interstitial leucocytic infiltration occurs in the vicinity of the affected tufts, while the tubules are only slightly affected. Most of the patients in whom blood-cultures have been positive have died, and only a few have recovered ; but it seems probable that a mild infection of the same kind is not uncommon, though blood-cultures are negative ; and that healing takes place, leaving, however, a chronic valvular defect and a chronic renal lesion.¹

The association between *aortic* valvular disease and renal disease is also striking, and has been recognized for many years. MacDonald² has examined *post-mortem* data upon this point and found that of 200 cases of renal fibrosis 34 (17 per cent.) had organic lesions of the aortic valve, while in 25 more the valves showed "compensatory thickening." Of 32 cases of aortic disease, 25 (78 per cent.) had renal fibrosis, which was present in 18 (81 per cent.) of 22 cases in which both aortic and mitral valves were diseased.

The varying relationships which exist between mitral and renal disease may also occur between the latter and aortic disease, but it seems probable that the renal lesion is less frequently secondary to aortic than to mitral disease.

4. *General Cardio-Vascular Degeneration.*—The influence of a generalized cardio-vascular degeneration in the production of chronic valvular disease has been in part considered in the preceding paragraphs. No clear distinction can be drawn between the heart and the arteries. The intimal lining is exactly similar, and the valves are merely a double layer of endothelium enclosing a delicate connective tissue ; and in cases of chronic arterial disease comparable lesions are often

¹ P. Baehr, *Amer. Journ. Med. Sci.*, 1912, cxliv., 317. J. F. Gaskell, *Journ. of Pathol.*, 1911-12, xvi., 287. E. Libman, *see* p. 229.

² MacDonald, D., *Glasgow Med. Journal*, 1914, lxxxi., 12.

found in the cusps. The aortic valve suffers most frequently, but atheromatous and calcareous plaques are not uncommonly found on the anterior mitral flap, and presumably own the same causes as the arterial disease. The mitral cusps, at any rate, possess arteries of their own, and any interference with their lumen will necessarily entail defective nutrition of the valves and produce lesions comparable to those which occur in the great vessels from such causes. (*See also* p. 217.)

5. *Congenital Defects.*—Congenital defects of the heart are not common, and though they generally involve the great vessels or the muscular septa, in many instances the valves as well are affected. The aortic and pulmonary cusps may thus be more or less numerous than usual, or represented by a diaphragm or a cone-shaped funnel. The orifice may be involved; one vessel may be narrowed and the other widened; or one alone may persist. The defects arise during the division of the *truncus arteriosus* into the pulmonary artery and the *aorta vera*. Many such cases have been recorded, but in Abbott's valuable article in Osler's "System" only four examples of developmental malformation of the mitral valve are recorded. It seems almost incredible that the auriculo-ventricular septum is so rarely abnormal, and much more probable that the nature of the defect has not been recognized. It is well known that valves which are congenitally defective are not infrequently attacked by acute endocarditis, and this may so disguise the abnormality that its real nature is not appreciated. A congenital defect may be the cause of some of the valvular lesions, which seem to own none of the more usual causes (*see* Chapter XXVII, p. 453).

The Actual Causes of Aortic and Mitral Disease.—The tables published by Lockhart Gillespie and by Grant Andrew, showing the age and sex incidence of chronic valvular disease, contain important indications as to its etiology, though the conclusions arrived at are only approximately correct, as the figures are those obtained at the time of admission to hospital, and show neither the age at which the disease originated nor the age at death, but merely that at which serious symptoms were present.

AGE.	AORTIC.						MITRAL.					
	Gillespie. ¹		Andrew. ¹		MacDonald.		Gillespie. ¹		Andrew. ¹		MacDonald.	
	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.	M.	F.
1 to 9	—	—	2	—	—	—	17	10	8	10	1	1
10 „ 19	13	2	22	5	6	—	78	114	76	118	8	10
20 „ 29	43	15	44	18	2	4	92	150	84	118	5	12
30 „ 39	66	14	65	17	11	2	106	96	93	96	7	12
40 „ 49	95	13	76	14	12	3	81	63	85	76	6	29
50 „ 59	126	13	48	8	13	—	210	86	75	37	1	9
60 „ 69			16	1	5	—			44	13	1	—
70	11	—			—	—	13	4	—	—	—	—
	354	57	273	63	49	9	597	523	465	468	29	73

The causes of aortic and mitral lesions are evidently different, for while the majority of aortic cases (56·3 per cent.) occur after the age of forty, the majority of mitral cases (61·3 per cent.) occur before that age; and while aortic disease is much more prevalent in men than in women, mitral disease affects the sexes in approximately equal proportions. The chief incidence of aortic disease in men is after forty; in women between twenty and fifty. Mitral disease is most common in men in the fourth decade, and in women in the third.

The causes which we have considered are likely to be active at particular periods of life. Developmental defects of course occur during intra-uterine life; acute endocarditis is most common in adolescence and in early adult life; syphilis and physical strain in early manhood; and high blood-pressure and cardio-vascular degeneration after middle life. Aortic disease in men thus seems likely to be mainly due to the influence of syphilis and physical strain; while in women acute endocarditis should be an important factor. Mitral disease in men seems likely to be mainly due to acute endocarditis and high blood-pressure and general degenerative changes; in women to acute endocarditis.

We have tried to elucidate the causes of chronic valvular

¹ Lockhart Gillespie, *Edin. Hosp. Reports*, 1898, v., 293. Andrew, J. G., *Age Incidence, Sex, and Comparative Frequency in Disease*, Lond., 1909, 156, 157.

disease by examination of our own records. David MacDonald¹ reported 58 cases of aortic valvular disease and 102 cases of mitral disease. J. K. Rennie² reported 104 cases of aortic valvular disease and 121 of mitral disease. One of us³ collected 70 cases of aortic valvular disease.

Congenital Defects.—Obvious congenital defects are not included in our series.

1. *Acute Endocarditis.*

Aortic Cases.—In MacDonald's series, 20 patients had suffered from rheumatism (34 per cent.). In 16 it was evidently acute rheumatism, but in the other cases its nature was indefinite. Sixteen other patients had suffered from acute infections of various kinds, and he considered that the incidence of acute endocarditis was not greater than 51 per cent., and was probably less. Rennie considered that 33.6 per cent. of his series were due to rheumatism.

Mitral Cases.—In MacDonald's series 68 patients had suffered from rheumatism (66 per cent.). Sixty patients had evidently suffered from acute rheumatism, 1 from gonorrheal rheumatism, the others from arthritis of unknown nature. Three patients had suffered from chorea, and 2 from recurring tonsillitis. In 25 other patients acute infections of various kinds were recorded. He considered that the incidence of acute endocarditis was not greater than 81 per cent., and in all probability less. Rennie considered that 69.4 per cent. of his cases were due to rheumatism.

2. *Syphilis.*

Aortic Cases.—In MacDonald's series, 10 patients (17 per cent.) had suffered from syphilis. In 10 cases aneurysm co-existed. These figures certainly unduly minimize the incidence of this disease. In Rennie's series the incidence was 30.7 per cent., in W.T.R.'s series 68 per cent. We strongly suspect that the last figures are those which most nearly represent the truth. Even on *post-mortem* examination it

¹ MacDonald, D., *Glasgow Med. Journal*, 1914, lxxxi., 12.

² Cowan, John, and Rennie, J. K., *B.M.J.*, 1921, ii., 184.

³ W. T. R. (unpublished). Males, non-syphilitic cases 18, syphilitic cases 48. Females, non-syphilitic cases 4, syphilitic cases 0. Total cases, 70.

may be difficult to establish the nature of a chronic valvular lesion, and it is still more difficult to do so during life. The history of past syphilis, a positive Wassermann reaction, the presence of other syphilitic stigmata, can alone establish the connection.¹

Mitral Cases.—In MacDonald's series, 1 patient had suffered from syphilis. Another patient had an aneurysm. In Rennie's series the incidence was 0·8 per cent.

The cause of 35·5 per cent. of the aortic cases, and of 29·7 per cent. of the mitral cases, in Rennie's series, was thus undetermined.

3. *Mechanical Strain.* — No examples of gross trauma occurred in the series. The influence of a continuously high blood-pressure, too, cannot be estimated in aortic disease, for in aortic regurgitation it occurs as a necessary result of the valvular disease, and independently of any associated defect elsewhere. In mitral disease a high blood-pressure is often present (36 out of 59 cases), but in many cases the evidence points to the elevation occurring subsequently to the onset of the cardiac lesion. In some cases, however, it seems probable that the connection is reversed, and that the valvular flaw is secondary to changes arising elsewhere. The association has already been considered at length on page 261.

4. *General Cardio-Vascular Degeneration.*—The evidence of vascular disease is not infrequently well marked in cases of chronic valvular affections. The brachial arteries are often notably tortuous, and the radials distinctly thickened, and one may be doubtful whether to catalogue a patient as a cardiac or an arterial case. In some the lesions may be very extensive.

A man, aged fifty-seven, was admitted into hospital complaining of a cough, accompanied by expectoration, and of great breathlessness which had begun five weeks previously, after a chill. The heart was considerably enlarged and double aortic murmurs were audible, as well

¹ In the *aortic* group only one syphilitic patient (3·1 per cent.) had mitral disease as well, while in the rheumatic group eighteen patients (54·2 per cent.) had also a mitral lesion. It is thus evident that the co-existence of mitral and aortic disease strongly suggests a rheumatic origin, while pure aortic disease suggests syphilis.

as that of mitral incompetence. The pulse was notably shotty. The whole of the arterial tree and most of the accessible veins were diseased. Radiograms disclosed a general dilatation of the aortic arch without any localized aneurysm ; and the temporal, facial, brachial, radial, ulnar, superficial volar, femoral, posterior tibial, and dorsalis pedis arteries were thickened, tortuous, and hard, though no calcification was apparent on X-ray examination. Ophthalmoscopic examination showed that the retinal arteries were normal.

The superficial veins of the legs were large and varicose, and the capillaries were dilated and visible halfway up the legs, the dilatation being maximal in the feet. The veins of the arms were large and tortuous ; and an anastomotic vein as large as a lead pencil crossed the hypogastrium from groin to groin, the flow of blood being from left to right.

The patient was a moulder by trade, and had been intemperate in early life, though for a number of years he had been an abstainer ; the Wassermann reaction was frankly positive. His symptoms were only cardiac in part. The right dorsalis pedis, though easily felt, did not pulsate, and several scars on the toes remained as evidence of gangrene which had occurred two years before admission. The œdema which was present on admission was trivial in degree and rapidly disappeared, and might quite well have resulted from the varicosity of the veins. Cheyne-Stokes breathing and a copious albuminuria showed that the kidneys were grossly diseased.

His early symptoms, however, were cardiac. Five years previously he had strained himself when at work, and had had to lie up for several weeks on account of breathlessness on exertion, œdema and præcordial pain ; and he had fainted repeatedly during the intervening years. During his convalescence in the ward he over-exerted himself on one occasion and the œdema recurred, and the liver and the heart became definitely enlarged, so that the cardiac reserve was evidently small. From the general point of view, however, the arterial mischief seemed the essential lesion, the aortic cusps being probably affected as a secondary result.

One must bear in mind that the presence of arterial disease does not necessarily indicate that the cardiac lesion is its result, for, as already mentioned, renal fibrosis and general arterio-sclerosis are not infrequently secondary effects of chronic valvular disease. The whole history of the case must be investigated and the evidence carefully weighed before a decision is made and a prognosis given. Cases of the primary arterial group are not far distant from their end when the cardiac valves have become incompetent, and they have, in consequence, a worse outlook than those cases in whom the valvular disease is primary. But in both definite arterial

damage is always serious, for it may lead to death rapidly and with but little warning.

Exact figures as to the incidence of vascular disease were unfortunately not available in MacDonald's series.

The special influence of mitral disease in the female sex between the ages of ten and thirty requires further consideration. It occurs at a period of life when acute endocarditis is most frequent, and is thus most likely to be due to rheumatic conditions. Grant Andrew's figures, however, show that acute rheumatism is more common in males than in females at those ages, and there is no evidence that endocarditis occurs more frequently in one sex than in the other during acute rheumatism. The explanation is probably to be found in its association with chorea, which is three times as common in girls as in boys.¹

Our records thus seem to support the conclusions which were deduced from more general consideration. Acute endocarditis of rheumatic origin is the most common cause of chronic mitral disease. Syphilis is the most important cause of chronic aortic disease, and then physical strain. Both aortic and mitral disease may occur in later life associated with chronic renal and arterial disease. The last group may arise in several ways; from high blood-pressure, from arterial lesions, or from the chronic intoxication which accompanies

¹ TABLE SHOWING THE AGE AND SEX INCIDENCE OF ACUTE RHEUMATISM AND CHOREA.

AGE.				ACUTE RHEUMATISM.				CHOREA.			
				Grant Andrew.		MacDonald.		Grant Andrew.		MacDonald.	
				M.	F.	M.	F.	M.	F.	M.	F.
1 to 9	..	..	..	7	5	2	2	11	45	2	5
10	..	19	..	77	82	23	23	46	134	11	27
20	..	29	..	132	95	31	18	1	17	—	2
30	..	39	..	69	32	19	12	4	—	—	—
40	..	49	..	24	19	6	4	—	—	—	—
50	..	59	..	13	3	1	1	—	—	—	—
60	..	..	..	2	—	—	—	—	—	—	—
				324	236	82	60	62	196	13	34

all chronic renal disease ; all three factors may be active at the same time.

A special group may be mentioned here. Chronic valvular disease is not uncommon in women who have borne many children, and is often accompanied by renal disease. It seems probable that in some instances the origin is to be ascribed to pregnancy producing coincident lesions in heart and kidneys.

In individual cases the cause of the valvular mischief is sometimes obscure, and all the usual factors may seem to be absent. An example was brought to our notice by a medical friend whose sister had been found, on a routine examination for insurance, to have well-marked mitral stenosis. Her age was twenty-four, and the most careful investigations failed to elicit any evidence of antecedent illness beyond a mild attack of measles. She had never suffered from rheumatism, tonsillitis, chorea, or scarlet fever, was little liable to "colds," and had always enjoyed excellent health. Her general development and nutrition were, if anything, above the average, and a developmental defect seemed the most probable explanation. Another patient, in whom mitral stenosis was discovered at the age of sixteen, had never suffered from any illness. In yet another patient, who was found to have well-marked aortic regurgitation at the age of nineteen, no cause could be determined.

CHAPTER XVII

THE SYMPTOMS OF CHRONIC VALVULAR DISEASE

THE symptoms of the various forms of chronic valvular disease differ in their incidence and their degree rather than in their nature, and may conveniently be considered together irrespective of the lesions with which they are most commonly associated. The special significance of particular symptoms will be considered subsequently.

The Causes of Symptoms.—Chronic valvular disease is not necessarily accompanied by symptoms of cardiac discomfort, and many individuals with valvular flaws are able to lead lives of average activity without distress. A valvular flaw, of course, imposes an extra strain upon the heart, but compensatory arrangements are made which overcome the particular difficulty. In aortic stenosis, for example, the lumen of the orifice is narrowed, and the flow of blood into the aorta is, in consequence, impeded. But if the left ventricle contracts more forcibly, and the rate of flow through the valve is sufficiently increased, the normal quantity of blood will enter the aorta in the normal period, and the disability will be overcome. Under ordinary circumstances, the heart works at less than half its full power, the reserve being retained to meet the extra strain of physical exertion; and any small degree of valvular stenosis or incompetence is readily compensated.

The additional work thrown upon the heart causes hypertrophy of the muscular fibres which are concerned, in exactly the same way that regular exercise causes hypertrophy of the systemic muscles; so that the additional work is carried on easily and well. But there is a limit to hypertrophy, and even when it is considerable, the power that can be developed

is *never so largely in excess of that ordinarily required* as in a normal heart. The reserve is only 30, 20, or 10 per cent., instead of the normal 50, and what Sir James Mackenzie calls "the field of response" is, in consequence, limited. A man may become breathless, for example, during a hard game of tennis, though able to walk about without discomfort. He may be able only to walk up a flight of stairs up which he could formerly run; he may only be able to walk quietly on the level; or he may be incapable of any exertion, though perfectly comfortable when seated on a chair. The difference is merely a matter of degree, but the response of the heart to demands for increased activity is more or less insufficient.

With a stationary lesion of moderate degree and good cardiac nutrition, the field of response may be large and ample, and the patient's disabilities trivial or imperceptible. Many individuals live for years without discomfort. Sir William Broadbent has recorded the case of a doctor, aged forty, in busy country practice, who had had aortic incompetence for twenty-six years. Sir William Osler reported the case of a lady, aged sixty-three, who had mitral regurgitation from the age of fifteen, but nevertheless possessed a healthy family of eleven children! Balfour knew a hale old gentleman of eighty-eight who had had a mitral leak for sixty-six years! And personally, we know two men, aged fifty-one and forty-one, with well-marked congenital defects, who have never suffered from any cardiac symptoms.

Many men with valvular disease served in the line during the recent war without difficulty. One of our patients, who had had acute endocarditis in 1909 affecting both aortic and mitral valves, was killed in action in 1918, after two years' service in France, without having suffered from any cardiac symptoms.

With a stationary lesion and ample compensation, symptoms are absent, and continue absent indefinitely; but in the majority of cases they make their appearance in course of time. Their incidence is generally due to the activity of some *new factor*, originating in the heart itself and affecting the valves or the muscle; or in some other viscus, and producing a disturbance of its action which indirectly affects the heart.

The valvular lesion may be *progressive* and the mechanical

difficulty may steadily increase. Congenital disease is as a rule associated with so great a disturbance of the circulation that it is incompatible with normal growth. Many of these children die shortly after birth ; some survive for a few years, but with stunted growth and imperfect nutrition, and readily succumb to accidental infections. But a congenital defect which has not interfered with normal growth or nutrition, and has not produced any symptoms before adult life is attained, is, in one respect, the valvular lesion which has the best prognosis, for it is one which is not progressive, and in which no recurrence of the causal factor can arise. The degenerative valvular lesions associated with syphilis or widespread arterial disease are, on the other hand, essentially progressive in their nature, and as years pass the valvular defect necessarily becomes greater, and the cardiac reserve smaller and smaller. The lesions which are due to acute endocarditis are intermediate in their tendency. A rheumatic scar contracts for a period of months or years, but not indefinitely, and an old-standing chronic lesion is, in consequence, stationary. But acute rheumatism tends to recur and is peculiarly apt to re-infect damaged valves, so that the cause may again come into action and the lesion may again become progressive. Muscular hypertrophy may attain to enormous size (a heart weighing 53 ounces has been recorded), but there is a limit to its development, and a progressive lesion in course of time produces mechanical difficulties which no attainable hypertrophy can compensate. With a progressive lesion cardiac failure sooner or later occurs.

The symptoms are often due to *failure of the cardiac muscle*. An individual's adaptability to his environment is always relative rather than absolute ; we cannot add a cubit to our stature ; and the most carefully regulated course of physical exercises may fail to produce an athlete from an individual who comes of poor stock. This vital factor is almost incalculable. A long-lived stock means a good family vitality, and the heart of a member of such a family is more likely to develop satisfactory hypertrophy, if it is required, than that of one belonging to a family whose members usually die in early life. Muscular failure arises in various ways, and may arise, too, in hearts which have no valvular defect.

The general nutrition of the patient is always a matter of importance, for the cardiac muscle shares in any general weakness. In mild cardiac failure a vicious circle is often established. Slight venous stasis occurs, the gastro-intestinal tract becomes congested, digestion and absorption become defective, the general nutrition and the cardiac nutrition are impaired, and the cardiac insufficiency is augmented.

The cardiac nutrition alone may be defective. This, too, is of common occurrence, for it is dependent almost wholly upon the integrity of the coronary arteries. And if, as is often the case, the blood-supply is lessened by narrowing of these arteries, at their origin or in their course, the cardiac reserve may be notably decreased.

A fatal termination is not infrequently due to a *fresh infection* of the valves, and *coincident myocarditis*. The valvular infection is often apparently trivial, a few small vegetations being scattered upon the surface of fibrous or calcified valves, though the myocardial inflammation is considerable.

A man, aged twenty-three, recently died under our observation. He had suffered from acute rheumatism in 1911, and was suffering from a second attack when he entered the wards for the first time in 1914. During the war he was regularly at work, but in the beginning of 1919 had to take a job which was more arduous than that at which he had worked during the war. Cardiac symptoms soon ensued, and he entered hospital again. By the end of the year he was able to walk four miles daily without discomfort, but in January of 1920 he caught cold and all his symptoms returned. At this time he had widespread bronchitis and double pleurisy, from which he made a good recovery, but he never regained his former vigour. The heart steadily dilated, and he died in May. There was no evidence during life of a fresh endocarditis. His temperature was generally normal, and only once touched 100·4° F. The leucocyte count was normal, and blood-cultures were sterile. But *post-mortem* examination showed a widespread myocarditis, mainly sub-acute but partly acute, associated with a few minute vegetations upon the mitral, aortic, and tricuspid valves, too small to have occasioned any gross valvular defect. The other cardiac lesions were all of old standing.

Disease of the coronary arteries occurs most commonly in association with syphilis and arterial degeneration. Myocarditis is most commonly associated with acute endocarditis. The former cause (coronary disease) is thus likely

to obtain in men, and after middle life. The latter (myocarditis) is most frequently met with in early life. Coronary disease is most frequently associated with aortic lesions, myocarditis with mitral disease.

Myocardial lesions not infrequently produce *alterations in the cardiac rhythm*, which in themselves throw an additional strain upon the heart. Heart-block, auricular flutter and fibrillation, nodal rhythm and even extrasystoles, may all occur. These disturbances in rhythm generally produce some signs of cardiac embarrassment, which are almost invariable in the case of fibrillation. The effects of disturbance of the rhythm are well seen in paroxysmal tachycardia, in which the heart dilates and the liver swells and œdema makes its appearance, if the paroxysm lasts for more than a few hours.

Extrinsic factors may come into action. These are innumerable, and may act in various ways. The records of athletics contain many examples of a heart strained by severe physical exertion, and even death may result, as in the Marathon race at Stockholm. Failure is more likely to occur in a heart if its reserve is lessened by chronic valvular disease, and the exertion need not be great or long-continued. In one of our patients the first symptoms of cardiac distress occurred during a run to catch a train; in another when lifting a pot in the kitchen; in a third when felling a tree. In cases of high blood-pressure, the pressure usually rises as years pass, and the extra strain one day becomes too great for the muscle to overtake.

The strain may fall upon the right ventricle instead of the left, from some affection of the lungs, bronchitis, pneumonia, or the like; and the strain may steadily increase, if, for instance, emphysema ensues. The right heart, it must be remembered, is already overworked in mitral disease and in affections of the pulmonary and tricuspid valves.

Symptoms may succeed a gastro-intestinal upset with its accompanying disturbance of nutrition and its varied intoxications; an operation; a mental shock; or starvation in any form. An acute exacerbation of chronic renal disease is frequently the last straw.

In another group symptoms ensue as a result of the cardiac

disease, but yet indirectly. An embolus may be set free from a clot in the auricles or on a valve, and a coronary artery may be blocked, or an infarct of the lungs or a cerebral softening may take place and cardiac failure follow. (*See* p. 304).

The Symptoms of Chronic Valvular Disease.—The most frequent and usually the first symptom in chronic valvular disease is *shortness of breath*, of which the majority of patients make more or less complaint. It may be frequency of respiration (polypæa), difficulty in breathing (dyspnœa), or associated with distress when lying down, so that the patient is forced to maintain the upright position (orthopnœa). Cheyne-Stokes or cyclic breathing may occur. The distress may be felt only on exertion, or during rest as well; and sometimes is most marked at night.

Shortness of breath occurs in many diseases. It may be due to anæmia or to disease of the lungs; it may be associated with abdominal conditions, such as the enlarged uterus of late pregnancy, which mechanically interfere with the free movements of the diaphragm; or may be the result of toxæmia. Comparable causes may be active in cardiac disease. Some patients are notably anæmic. The weakness of the left heart may be so great that systemic ischæmia ensues on even trivial exertion. The lungs are often more or less engorged and pulmonary œdema is the rule if the failure is well marked; bronchitis and emphysema often co-exist; and an infarct or pleural effusion may add to the respiratory difficulties. An enlarged liver or ascites may hinder the movements of the diaphragm. Associated renal or hepatic disease may produce toxæmia.

It is important to recognize in each individual case the particular factor that is in action, and this is usually easy if a routine examination is made, for the signs of bronchitis, pulmonary œdema, ascites, etc., are distinctive. Toxic causes may occasion difficulty, for chronic renal disease of the cirrhotic type is not invariably accompanied by albuminuria; which, conversely, may be the result merely of chronic venous congestion. The blood-pressure is here a useful guide, for it is not elevated in simple valvular disease, save in cases of aortic incompetence, so that a high blood-pressure

indicates the existence of some extra-cardiac complication, such as arterial or renal disease, and most frequently, defective elimination. The character of the respiratory distress is important, for paroxysmal dyspnœa is always toxic in origin; while orthopnœa, in the absence of gross pulmonary or abdominal mischief, owns a similar cause.

In the typical form of *Cheyne-Stokes breathing* periods of apnœa and dyspnœa alternate. The respirations, at first weak and infrequent, gradually become deeper and more numerous, and then gradually wane until respiration wholly ceases. The alternation is sometimes incomplete, and no real apnœa occurs, though periods of weak infrequent breathing alternate with periods during which it is full and frequent. Similar variations may occur in the pulse rate, which is generally more rapid during apnœa, but the respiratory and pulse variations do not coincide exactly, though they exhibit similar curves (Fig. 193); and the relationship may be reversed, the pulse rate being more frequent during dyspnœa; it is often unchanged (Fig. 194). A "pulsus alternans" may occur during dyspnœa, if the respirations number half the pulse rate (Fig. 195); the smaller beat occurs at the maximum of inspiration. (See also Fig. 132, p. 149.)

The periods of apnœa vary in duration and may last for a second or two, or for nearly half a minute or even longer. If the period is long, the patient often becomes unconscious and may close his eyes and cease talking, individual muscles may twitch, and, rarely, general convulsions occur. In the lesser degrees the intelligence may be unimpaired. If the eyelids remain open, the size of the pupils may be seen to vary; they usually contract during apnœa, and dilate during dyspnœa; but the phenomenon is inconstant and variable.

Cheyne-Stokes breathing is most frequently noticed at night, though it may be continuous; and in many cases the patient is greatly distressed, for as soon as he goes to sleep the periodicity is exaggerated, apnœa is prolonged, and at the end of each period he awakens up in distress, covered with profuse perspiration, and may have to sit up in bed to recover his breath. In the lesser grades symptoms may be absent. In some cases the breathing is periodic or cyclic, the waxing and waning of the typical Cheyne-Stokes paroxysm being

absent, the patient merely ceasing to breathe for a few seconds at more or less irregular intervals.

Cheyne-Stokes and cyclic breathing occur in cerebral disease as well as in toxic conditions. Cushing¹ has shown experimentally that an increase of intracranial pressure produces Traube-Hering waves of blood-pressure, which are associated

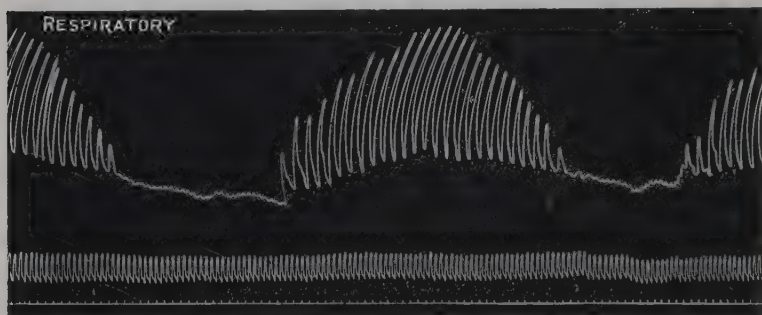


FIG. 194.—CHEYNE-STOKES BREATHING. SIMULTANEOUS RESPIRATORY AND PULSE TRACINGS.

The pulse rate remains unaltered throughout. The time record below the arterial tracing is one-fifth of a second.

with periodic breathing. The fall of blood-pressure, with its resulting cerebral anæmia, is accompanied by apnœa and slowing of the pulse rate. The rise of pressure, with re-establishment of the cerebral circulation, is accompanied by hyperpnœa and increase in the pulse rate.

Eyster² has pointed out that, clinically, there are two



FIG. 195.—BRACHIAL (UPPER) AND RESPIRATORY (LOWER) CURVES SHOWING PULSUS PARADOXUS WHICH SIMULATES PULSUS ALTERNANS.

types of Cheyne-Stokes breathing. In one, the cerebral group, the blood-pressure and pulse rate rise during hyperpnœa. In the other the blood-pressure and pulse rate fall (Fig. 193). The latter group obtain in renal and cardio-

¹ Fulton, F. T., *Heart*, 1915, vi., 77.

² Eyster, J. A. E., *Jour. Exper. Med.*, 1906, viii., 565.

vascular cases, presumably from toxic causes (*see* p. 27). We have observed both of these types, and also a third in which no alteration of the blood-pressure or pulse rate can be detected (Fig. 194). The lesions in chronic cardiac disease are, as has already been mentioned, often widespread and not confined to the heart alone, cerebral, pulmonary, and renal conditions co-existing. One or other, in any particular case, may dominate the picture, but all may be present, with, in consequence, a varying result. The typical case is, in our experience, the exception.

Paroxysmal attacks of breathlessness occasionally occur. They are most common at night, the patient as a rule suddenly awakening from sleep in an access of intense dyspnœa, which necessitates his sitting bolt upright in bed. Expiration is prolonged, and the chest is full of rhonchi. After an interval of some duration (minutes to hours) relief is obtained, but some orthopnœa generally persists. The attacks tend to recur, and the distress may be so great that the patients are afraid to go to sleep.

Cough and expectoration are not uncommon. The cough is usually accompanied by expectoration, and due to a slight bronchial catarrh, or œdema of the lungs; but it is sometimes an isolated symptom and may be extremely troublesome. The sputum may be of varied kind; mucoid or mucopurulent from catarrh; watery and perhaps copious in œdema of the lungs, when a spittoonful or more may be expectorated in twenty-four hours; or hæmorrhagic. Hæmoptysis in chronic mitral disease is very seldom due to pulmonary phthisis. In mitral stenosis hæmorrhage occasionally occurs from capillary rupture when the congestion of the vessels is extreme; and several ounces may be voided. As a rule it ceases quickly, though it may recur, and the patient is subsequently conscious of improvement in his general condition. In one of our patients, a girl who had been sent to a sanatorium owing to an error in diagnosis, the relief was so distinct that she welcomed recurrence of the hæmorrhage. In the other valvular lesions simple rupture is unusual, and the most common cause of hæmoptysis is a pulmonary infarct. The bleeding in this case is not accompanied by relief, but rather by an exaggeration of the symptoms, and the breathlessness

may be intensified suddenly and accompanied by pain in the side from associated pleurisy. The physical signs of a consolidation are sometimes apparent on examination. The prognosis is always serious, for a pulmonary infarct is evidence as a rule of gross stasis in the right auricle, and is thus the accompaniment of a considerable failure of compensation, and is likely to occur towards the end of the illness; but in exceptional cases a fair recovery may ensue and persist.

Cough without expectoration is not common. Paralysis of the left, rarely of the right, recurrent laryngeal nerve, associated with mitral stenosis, has been recorded in seventy cases.¹ The paralysis is probably due to compression of the nerve between the left pulmonary artery and the aorta or aortic ligament,² and not to direct compression by the left auricle.

Cardiac discomfort or pain is frequently experienced. The most common of these complaints is palpitation, the patient becoming conscious of the cardiac contractions, which he describes as fluttering, bumping, or throbbing, felt in the cardiac region, the neck or the head.

Palpitation, of course, may occur in healthy individuals under the stress of severe physical exertion, and is then an indication that the cardiac work is excessive and must be lessened. A similar origin may be apparent in our patients, the discomfort arising during exertion and ceasing during rest. But the degree of exertion which produces it is slight, and it obtains upon exertion which in the past produced no distress. The heart for some reason has become less able to cope with extraordinary demands upon it, its reserve power is lessened. In these cases there is usually some other evidence of cardiac insufficiency, a frequent pulse rate, an enlarged or tender liver, or some œdema in the feet at night. Palpitation of this kind is most common in patients with large hearts, in particular those associated with arterio-sclerosis, renal disease, and aortic regurgitation. In the latter case the cause is obvious. One can often see the head shaking with each ventricular systole, and we have even felt the bed on which the patient was lying vibrate in unison with the heart. The

¹ Garland, J., and White, P. D., *Arch. of Int. Med.*, 1920, xxvi., 343.

² Fetterolf, G., and Norris, G. W., *Amer. Journ. of Med. Sci.*, 1911, cxli., 625.

sudden variations in pressure which occur within the vessels occur also within the heart, and from their vehemence convey impressions to its walls which are transmitted to the brain.

In other instances palpitation occurs during rest, not infrequently at night. One of the most common causes of this group is flatulent distension of the stomach. The heart is extremely mobile. The apex impulse moves an inch or more if one turns over on to the left side; and the organ follows the diaphragm in its excursions, which, as X-rays show, are considerable. The stomach lies immediately beneath the diaphragm and, if distended, presses upwards against the heart and may incommode its action to such an extent that this becomes obvious to the patient. The heart is not at fault, and the prognosis and the treatment depend upon the character of the gastric disorder.

In another group the cause is to be found in disturbance of the cardiac rhythm. The occurrence of an extrasystole may thus be appreciated, the patient being conscious of the long pause following it, or of the strong post-extrasystolic beat. More rarely the extrasystole itself is felt. The post-extrasystolic beat is appreciated on account of the ventricle being more completely filled than usual from the prolongation of diastole, so that the heart is overtaxed, while the arteries are more completely filled. The extrasystole may be perceived if auricle and ventricle contract simultaneously (Fig. 83), so that the auricle is unable to empty itself through the shut auriculo-ventricular valve. One can sometimes feel the shock when listening over the heart with a wooden stethoscope. But in many cases the coincidence of the auricular and ventricular contraction is not exact, and if the post-extrasystolic pause is short, the patient may be unaware of the irregularity.

Palpitation is a common complaint in auricular flutter, fibrillation, nodal rhythm and heart-block. In fibrillation the output of the ventricle is very variable, and both the amount of cardiac work and the distension of the arteries vary with successive beats. In the others, the shock of the auricular contraction may be appreciated if the auriculo-ventricular valve is shut. The palpitation of tachycardia in such diseases as exophthalmic goitre may own a similar

cause, for if the pulse rate is very frequent ventricular diastole is short, and auricular contraction may occur before the preceding ventricular contraction has ended (Fig. 66).

Irregularity of the heart is, however, by no means always appreciated, and another factor, an increased sensibility to internal stimuli, must consequently often be in operation. Emotion, for example, is frequently accompanied by palpitation without any undue frequency or irregularity of pulse, and palpitation is more common in nervous individuals than in those of phlegmatic type. The sole factor is sometimes merely this hypersensitiveness, but a careful examination of the heart should invariably be made before a cardiac origin is definitely excluded.

Palpitation is most common in cases where the cardiac action is frequent, and is comparatively rare when the pulse rate is below normal. In heart-block, however, when auricle and ventricle happen to contract simultaneously, the shock may be appreciated, though the pulse rate is infrequent.

Pain may be occasioned in several ways and may be referred to various situations. Epigastric pain of a dull, aching character, intensified by pressure and often by the ingestion of food, is usually associated with a large congested liver, which may be felt upon palpation. Epigastric pain is rarely due to cholecystitis, which occurred in a patient who was recently under our care for severe cardiac failure. Embolism often occasions abdominal pain, which may be very severe. The most common cause is embolism of the spleen, in which case the pain is generally referred to the left side, where friction may be audible on auscultation; but the pain may be general. A woman, aged forty-six, who had suffered from symptoms of mild cardiac failure for a couple of years, was one day seized, without obvious cause, with griping abdominal pain, which was so severe that she fell down upon the floor and was unable to move until her husband came home from work five hours later and lifted her into bed. The only obvious cause on *post-mortem* examination was a splenic infarct. In another patient who was already in hospital, abdominal pain of severe character suddenly ensued when he was lying quietly in bed, and required the administration of morphia; the stools on the next day contained blood in large amount. *Post-mortem*

examination revealed a blocked mesenteric artery with necrosis of several feet of the small bowel, and general peritonitis.

Thoracic pain also owns several causes. It may be due to pulmonary infarct and pleurisy, and is then usually referred to the side of the chest. It may be due to pericarditis, and is then præcordial in site. It may be paroxysmal and of anginous type, præcordial in site, but often spreading to the head and arms (*see* p. 388). A clear distinction should be drawn between continuous and paroxysmal pain, for the former is almost always associated with a gross visceral lesion, while the latter *may* be due to functional disturbance.

The degree of thoracic discomfort varies considerably in different patients. In some the complaint is frequent, but the distress is probably trivial, and no objective evidence of cardiac weakness may be apparent. A choking sensation, a short bout of palpitation, or a sense of constriction round the chest, may thus be called pain by some individuals, while in others little reference is made to discomfort and complaint is made of pain alone.

Cardiac œdema contrasts with that of renal disease in being most marked in the lower extremities, and first noticed in the evening, if the patient has been walking about during the day; and minimal in the morning, after he has been lying flat in bed. Œdema may be considerable without attracting the attention of the patient. The rapid variations in the weight of cardiac patients from time to time are generally due to fluctuations in the amount of fluid in the subcutaneous tissue, and a stone's weight may be present without any very obvious signs. In patients who are confined to bed, the sacrum and the back of the thighs may be œdematous, as they are the most dependent parts, though the shins scarcely pit.

The œdema spreads upwards and may involve the scrotum and the serous sacs. Œdema of the prepuce may interfere with micturition; the difficulty can always be temporarily overcome by firm pressure on the foreskin with the fingers. Pleural effusions, on the right side more frequently than on the left, are perhaps more common than ascites, but ascites may be missed if the patient is orthopnœic, for the pelvis can contain a considerable quantity of fluid without any obvious signs of its presence. Pleural effusion, on the other hand, may

occasion considerable respiratory distress even when the amount is small.

In a general way the œdema and effusions steadily increase from below upwards, and any deviation from the rule indicates the activity of some new factor. Pleural adhesion, for instance, may prevent pleural transudation, and should be suspected if one pleural cavity is full and the other empty; and the presence of gross ascites, if its degree is not proportionate to that of the general œdema, suggests the presence of hepatic cirrhosis. In chronic venous congestion, the liver is always large, and the absence of its enlargement in cardiac failure indicates the presence of some hepatic cause. In a case of adherent pericardium a correct diagnosis was suggested by consideration of these factors. The patient had recently suffered from acute rheumatism, but was admitted to hospital on account of ascites. The liver was enlarged, but œdema was absent, and the cardiac action was in no way disturbed, which suggested the diagnosis of adherent pericarditis and hepatic fibrosis, a diagnosis which was verified on *post-mortem* examination eighteen months afterwards.

Gastro-intestinal symptoms are frequently present. They are generally the result of chronic venous congestion of the alimentary tract, and their occurrence, interfering with the proper nutrition of the patient, or increasing his discomforts and weakness, adds largely to the gravity of the prognosis. Anorexia, flatulence, nausea, and vomiting may all occur. There may be discomfort or pain after food. Of intestinal symptoms flatulence is the most common, and by its interference with the movements of the diaphragm may interfere with respiration. Constipation is the rule, but a watery diarrhœa sometimes ensues. Hæmatemesis and melæna are unusual, and, if severe, are generally due to associated hepatic disease, but ulceration may occur in any part of the alimentary tract. Catarrhal conditions are extremely likely to occur in chronic venous congestion from even trivial dietetic indiscretions, and their occurrence necessarily increases the severity of the symptoms.

In failure of the right heart the *liver* is congested and enlarged, and is tender on palpation. Spontaneous pain, too, may be experienced; it is sometimes constant, but is more

usually related to digestion, and occurs from one to three hours after meals. In these cases the liver can be felt to project beneath the right costal margin. The surface is smooth and the edge is sharp and clearly defined, and is everywhere tender on palpation. In chronic cases the enlargement may be extreme and the lower edge may be situated below the level of the umbilicus. The hepatic veins in these cases are often greatly dilated, and *post-mortem* may accommodate the little finger as high as the first joint, and true hepatic pulsation may be detected during life. The liver may pulsate from many causes. The pulsation may be communicated from the aorta, or through the diaphragm from a hypertrophied right heart, but neither of these pulsations are *expansile*. When the tricuspid valve is incompetent and the contents of the right ventricle are sent in part into the veins with each ventricular systole, the hands of an observer placed beneath the false ribs, and in front just below the costal margin, can be felt to separate from each other, which cannot occur in mere communicated pulsation, as the liver is cone-shaped and the apex is the highest point. The liver may be displaced downwards if the right pleural sac contains fluid, and enlargement may be simulated; the condition can be recognized by careful examination.

Jaundice is not uncommon. Its occurrence is always of serious significance, for it generally appears in the later stages of cardiac failure and is not infrequently a terminal event. It is usually associated with gastro-intestinal catarrh, to which the catarrh of the hepatic ducts must be related; but the hepatic congestion predisposes to its occurrence. Cholecystitis may occur, but it is rare in our experience, though gall-stones are often found in cardiac cases. Brockbank's experience may be quoted. In his series (*post-mortem*) gall-stones were found in 55 cases (10 per cent.) out of 504 cases of gross cardiac disease, while they only occurred in 46 instances (5 per cent.) out of 843 cases which had no cardiac disease. At St. George's Hospital Mills found that gall-stones were present in 30 (15 per cent.) out of 200 cases of cardiac disease.¹

Cerebral symptoms are often present. They may be due to gross cerebral lesions of arterial origin, from rupture, or

¹ Rolleston, H. D., *Diseases of the Liver*, Lond., 1905.

thrombotic or embolic blocking, but may be merely a symptom of the cardiac failure. Faintness, partial or complete, sometimes momentary, and in other instances of longer duration, attacks of giddiness, headache, insomnia, a sense of impending dissolution, may all occur. Many patients complain of constant throbbing in the head; this is most frequently associated with aortic incompetence. Headache and insomnia are often accompanied by paroxysmal dyspnoea or by orthopnoea, and are usually due to toxæmia and associated with a high blood-pressure. Mental symptoms may obtain; patients with nocturnal dyspnoea often suffer from disturbing dreams, which may appear as real occurrences during their waking hours; and may occasion distressing difficulties with their attendants; or the patients may be mildly delirious or stupid. Acute mania is rare; dementia is not uncommon, but is most usually associated with evidence of organic cerebral disease.

The *urine* is frequently abnormal. In cardiac failure it is scanty in amount, high-coloured, of high specific gravity, very acid, and may contain albumen in considerable quantity, even in the absence of renal disease. In these patients micturition may be frequent, and tenesmus troublesome, interfering with sleep. With improvement the output returns to normal.

It is always important to determine, if possible, whether the dropsy and albuminuria are due to venous congestion of the kidneys or whether there is coincident sub-acute or chronic nephritis. In solving this problem all the clinical manifestations must be taken into consideration and regard must be had to the presence or absence of tube casts in the urine. Further help may be afforded by the urea concentration test.¹ The patient, having emptied the bladder, is given 15 grammes of urea dissolved in 100 c.c. of water. One hour and two hours later, the patient empties the bladder, and the percentage of urea in the two specimens is estimated. The specimen passed at the end of the second hour is the more important. If it contains 2·5 per cent. of urea the functional efficiency of the kidneys is satisfactory. In nephritis, whether acute, sub-acute, or chronic, we may find a normal percentage of urea. Thus in a woman aged twenty-

¹ Maclean, H., *Brit. Med. Journ.*, 1921, ii., 425.

four, who had been suffering from sub-acute nephritis for eighteen months, the percentage was 2.6. In others it is low, for example, 1.4 in a case of five years' duration, and 0.6 in another case of six years' duration. In cardiac failure the percentage may be normal, especially if there be no dropsy. Thus in a man aged sixty-three, suffering from auricular fibrillation of syphilitic origin, who had presented dropsy and albuminuria, and who was still very breathless on the least exertion, the urea concentration test gave a percentage of 2.9.

In many cases of cardiac failure, however, the percentage of urea is below normal, as in the case of a woman aged fifty, suffering from mitral incompetence and auricular fibrillation of rheumatic origin, with intermittent dropsy for five years. In this case the urea concentration test gave a percentage of only 2.1. In determining the efficiency of the kidneys, use may also be made of the phenol-sulphonephthalein test and of blood-urea estimations. Hæmaturia is probably always absent in simple venous congestion, but is of common occurrence in cases where the kidneys are also diseased. It is sometimes very persistent in those cases which are complicated by renal fibrosis. Hæmaturia also occurs from renal embolism, but this is more common in acute than in chronic valvular disease.

The Symptoms of Mitral Disease.—It is impossible to draw a sharp clinical distinction between mitral stenosis and mitral incompetence. Their causes are similar—acute endocarditis in early life and chronic endocarditis in later years—and they are frequently associated with chronic arterial and renal disease. Incompetence, however, owns one cause which is peculiar to itself. The orifice of the mitral valve is large, while the cusps are relatively small and quite incapable of closing the aperture unless it is narrowed by the contraction of the sphincter muscle. In anæmia and in debility from any cause, this sphincter action may fail, and a “functional” mitral incompetence ensue. A comparable defect may occur also in cases of aortic valvular disease or high blood-pressure, where a special strain is thrown upon the left ventricle. These “functional” regurgitations are usually of short duration, but one of our patients who had a transitory ventriculo-

systolic murmur at the apex at times in 1911 and 1912, had the murmur permanently present after 1915 until his death in 1920, though the *post-mortem* examination showed the absence of organic valvular disease.

The presystolic murmur is *always* a sign of organic valvular disease, but the systolic murmur may be due to a temporary and curable cause.

In the majority of cases of organic valvular disease stenosis and incompetence co-exist, though the degree of each varies in individual cases, the stenosis being sometimes considerable and the incompetence trivial; and *vice versa*. In many cases a double murmur is present, but in others one alone persists, and an accurate diagnosis may be impossible during life. Thus, in auricular fibrillation, the characteristic crescendo murmur disappears with the cessation of co-ordinate auricular contraction; and the murmur also fails when, as in nodal rhythm and heart-block, the auricular contractions do not immediately precede the ventricular contractions. The presystolic murmur may alone obtain in cases where *post-mortem* the mitral orifice is found to be patent, and the cusps so stiff and hard that it seems impossible that the valve could ever close. Regurgitation in these cases is prevented by the high blood-pressure within the auricle, which is raised by the contraction of the right ventricle to such a height that blood can only flow slowly, if at all, from left ventricle to left auricle during ventricular systole, and too slowly to produce an audible murmur. The presystolic murmur is the one that most often disappears, and our experience agrees with that of Graham Steell,¹ who states that the murmur most frequently present in cases of mitral stenosis is unquestionably the murmur of coincident incompetence.

In both lesions the pulmonary blood-pressure is high. In stenosis the elevation is continuous, for the obstruction at the mitral valve necessarily remains constant. In incompetence, on the other hand, the elevation is intermittent, for though the left auricle is distended when the ventricles are in systole, by blood flowing into it from the left ventricle and the pulmonary veins, the congestion is rapidly relieved when diastole succeeds, as no obstacle is now presented to the free

¹ *Diseases of the Heart*, Manchester, 1906, 121.

flow of blood into the left ventricle ; and the auricular blood-pressure in consequence rapidly falls. For this reason, dilatation of the left auricle is as a rule more extreme in cases of stenosis, and clots in the auricles are more common, and embolic phenomena more frequent.

In mitral incompetence the left ventricle is dilated and hypertrophied. During ventricular systole the left auricle is distended by blood flowing into it from the left ventricle and the pulmonary veins, and the left ventricle, in consequence, receives during its diastole a larger amount of blood than normal. The larger complement, too, is necessary if compensation is to be sufficient, for a normal amount of blood in the left ventricle would be insufficient to supply the aorta with its proper amount, if some of the blood regurgitated at the same time into the left auricle. Mitral stenosis, on the other hand, has no effect upon the left ventricle, but only on the left auricle and the right ventricle ; and left ventricular hypertrophy, when it obtains in this lesion, is *due to other causes*, aortic valvular, arterial, or renal disease.

The *onset of symptoms* may be gradual or abrupt. The slighter symptoms are often insidious, but those which bring the patient under observation as a rule ensue more or less suddenly. Thus in MacDonald's series¹ the onset was gradual in 36 cases (35·2 per cent.) and more or less sudden in 66 (64·6 per cent.).

In the exceptional case the symptoms may arise with dramatic abruptness. A woman, aged forty-three, had experienced slight breathlessness and palpitation on exertion for some years, but of so trivial a degree that she paid no attention to the discomfort and sought no advice. One morning her husband, on awakening, discovered that she was unable to speak and could not move the right arm or leg. On *post-mortem* examination a large cortical softening was found in the left cerebral hemisphere ; and well-marked mitral stenosis. The embolus was evidently derived from clots in the left auricle. Such a course of events is, however, uncommon, but serious symptoms may arise within a few days. A man, aged forty-six, a foreman with a firm of horse-dealers, took a shipload of horses to Canada for sale. On

¹ MacDonald, D., *Glasgow Med. Journ.*, 1914, lxxxi., 12.

September 21st he ran the horses in the show-yard without discomfort. He immediately sailed for home, but on the voyage bad weather was encountered, and on the 27th his cabin was flooded. Bronchitis ensued, and two days later his legs were œdematous. On admission to hospital the cardiac failure was extreme, but he made a good recovery. The bronchitis had overtaxed a right heart already strained by a double mitral lesion. A woman, aged thirty-five, who had always been healthy save for an attack of acute rheumatism at the age of twenty, noticed that her feet were swollen after the sixth month of her fourth pregnancy. Labour was difficult and prolonged, and chloroform was administered; she lost a considerable quantity of blood. The œdema rapidly disappeared, and she felt quite well when she rose from bed on the ninth day after delivery, but the swelling returned within a week, and steadily increased in amount, and she became very short of breath, even while at rest. The failure continued, and she died some months afterwards. The strain of pregnancy and a post-hæmorrhagic anæmia had occasioned the failure of compensation.

The onset may be gradual. A little wizened old woman enters hospital every few months suffering from extreme general weakness and considerable œdema. She has a well-marked presystolic murmur. Improvement is always rapid, and in a month or six weeks she has gained a stone in weight and feels in excellent health. But she drinks heavily, and after a few weeks invariably loses her employment and is half starved; and the symptoms slowly recur.

The onset of symptoms may be determined by various causes. Twenty-two (21·5 per cent.) of MacDonald's cases were admitted to hospital on account of other diseases (acute rheumatism, 11; influenza, 6; scarlatina, gonococcal arthritis, pleurisy, gastritis, ruptured pyloric ulcer, each 1). In 13 cases (12·7 per cent.) symptoms followed exposure to cold and chill, and in 10 cases (9·8 per cent.) childbirth, and in 2 more arose during pregnancy; in 5 (4·9 per cent.) they succeeded exertion (a "flitting," a game of football, a dance). Starvation, angina pectoris, the shock of a husband's illness and death, the extraction of several teeth, etc., are included among the causal factors in his list.

The Symptoms of Aortic Disease.—In aortic, as in mitral disease, it is impossible to draw a sharp distinction between stenosis and incompetence, for the two lesions frequently co-exist, though the degree of each varies in individual cases. In a general way incompetence is much more common than stenosis, and the more serious fault; but, whenever the cusps are stiffened, their mobility is lessened and the orifice is slightly narrowed, and double murmurs are, in consequence, audible; even trivial vegetations, too, may produce a systolic murmur.

The diastolic murmur is almost always a sign of organic disease of the valve, or at any rate of permanent incompetence, for though it may occur without any lesions of the cusps from dilatation of the aortic orifice, the connective and elastic tissues of the wall, when once stretched, never regain their former condition.

Cases of aortic incompetence without valvular disease are uncommon, but George Middleton recently showed one of us a specimen from a patient who had died in his wards. The valve was grossly incompetent to the water-test, but the cusps were only slightly thickened and were not retracted, though they were quite incapable of closing the greatly dilated orifice. The ascending aorta, too, was dilated, the walls being affected by an advanced syphilitic aortitis.

In a few rare cases, however, the murmur may be inconstant, and this may occur both in acute endocarditis and in chronic degenerative lesions. In the former case large vegetations may temporarily stop the leak; in the latter the leak may be minimal for a time. The loudness of a murmur depends upon two factors, the rapidity of the blood-current and the nature of the valvular fault; and may vary from variations in the first factor, even though the second remains constant. An old man, who was attending the out-patient clinique, had at one time a diastolic murmur which was only audible immediately after exertion; it disappeared after ten minutes' rest. In the course of some months the murmur became constantly present, and as the other symptoms increased he was admitted into hospital and put to bed. Two days later the murmur was absent so long as he was in the recumbent position, but reappeared whenever he stood erect.

With physical exercise the blood-pressure rises, and the difference between the blood-pressures within the aorta and the left ventricle during ventricular diastole is, in consequence, increased. The backward flow may now be sufficiently rapid to produce an audible murmur, though during rest the pressures approximate, the rate of flow lessens, and the murmur disappears. In this patient, as the incompetence progressed, the murmur became audible whenever gravity was in action and the difference in pressure was augmented, though it was absent when the patient was recumbent.

An inconstant murmur as a rule in time becomes constant, but a few cases have been recorded in which the murmur has never returned. The explanation is difficult to understand. In a still more rare group the diastolic murmur may be absent, though *post-mortem* the valve is stenosed, and the cusps so stiff and hard that it seems certain that the orifice was always open, while to the water-test it is markedly incompetent.

The normal blood-pressure within the aorta is considerable (100 to 130 mm. Hg), while the aortic cusps are thin and fragile, and it has been suggested that they are of themselves insufficient to prevent leakage unless supported on the ventricular side by the apposition of the subjacent muscle. If the aortic bulb is dilated, the support fails, and regurgitation ensues. With recovery the muscular pads resume their function, and the leak is stayed; while conversely, in valvular disease the muscular mechanism may be sufficient to prevent regurgitation. Keith considers that though the subaortic musculature does contract and narrow the lumen during the systole of the ventricles, its contraction ceases during diastole, and that it can have little if any effect in preventing regurgitation at this period of the cardiac cycle. Stewart,¹ however, maintains that the musculature of the aortic conus enters into contraction later than the rest of the ventricular muscle, and remains contracted during the early part of diastole, thus affording the aortic cusps efficient support until the intra-ventricular pressure rises, with the influx of blood from the left auricle, to such a height that regurgitation can only be minimal.

The systolic aortic murmur, on the other hand, is by no

¹ Stewart, H. A., *Arch. Int. Med.*, 1908, i., 102.

means definite evidence of aortic stenosis. It is often present when the cusps are merely hard and stiff or coated with small vegetations in such trivial degree that little obstruction can obtain; and frequently occurs in anæmia or debility, disappearing as soon as the general condition is improved. In fact, as an isolated sign, it is more frequently associated with a normal than with an abnormal valve.

The effects of aortic valvular disease are at first confined to the systemic circulation and to the left ventricle. In stenosis the lesion hinders the entrance of blood into the aorta and tends to produce systemic anæmia; in incompetence the blood enters the aorta readily during systole, but re-enters the ventricle as soon as diastole succeeds; the systemic anæmia is intermittent. In stenosis the lesion in itself tends to lower the blood-pressure, though, as a matter of fact, it is generally high from associated arterial and renal disease. In incompetence the blood-pressure is necessarily elevated as a result of the incompetence; for if the mean blood-pressure within the aorta is to be maintained at normal levels, the initial pressure must be above the normal to compensate the rapid fall which occurs during the early part of diastole. The variations in pressure may be large if the leak is great, and the pulse-pressure (the difference between the systolic and diastolic pressures) is an accurate index of the degree of the mechanical fault (Fig. 196). Some of the symptoms—the throbbing in the head, palpitation, etc.—are the result of this sudden variation of the arterial blood-pressure.

Aortic stenosis is compensated by ventricular hypertrophy, but in incompetence the ventricle must be larger than normal as well as hypertrophied; for if the normal amount of blood is to be left in the arteries at the end of diastole, the amount thrown into them during systole must be greater than normal to compensate the loss which occurs during diastole. In aortic incompetence the left ventricle may be very large. The heart of one of our patients who suffered from double aortic disease weighed 40 ounces.

The strain of aortic lesions is thus thrown primarily upon the left ventricle, and as long as it is able to do its work, symptoms are in abeyance. But if the work required is more than it can undertake, it dilates under the strain, the sphincter

muscle of the mitral valve fails to close the orifice, mitral regurgitation occurs, and the symptoms of an engorged pulmonary circulation are superadded to those of the original lesion. In the majority of aortic cases the onset of serious symptoms is synchronous with the onset of mitral incom-

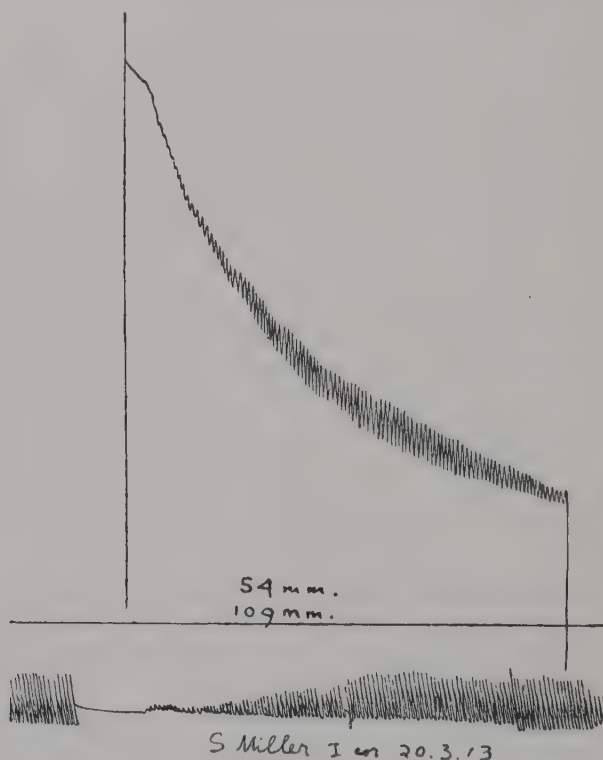


FIG. 196.—BLOOD PRESSURE CHART. AORTIC INCOMPETENCE.

The pulse pressure (55 mm. Hg.) exceeds the diastolic pressure. Note the large shotty pulse, as recorded in the sphygmographic tracing in the lower part of the illustration.

petence, and in great measure its result, and aortic and mitral disease thus produce symptoms of the same type, though they vary in their incidence and in their degree. In mitral disease symptoms ensue when the right heart fails; in aortic disease when the left heart fails.

Aortic disease is associated with arterial and renal lesions with even greater frequency than is the case in mitral disease, and the symptoms which obtain are often only partly due to the cardiac element.

The *onset of symptoms* may be gradual or sudden, but is most often the former, being sudden in only 15 (25·8 per cent.) of MacDonald's series, and gradual in 43 (73·9 per cent.). In some cases they succeed physical exertion, as was the case with a man, aged fifty-three, in active business, whose aortic valves were known to have been incompetent for two years, though no evidence of cardiac weakness had been shown; and who had a serious faint as the initial symptom after a hard day's work in hot weather. In another case, an engineer aged twenty-five, whose valvular disease had been recognized for several years but had not interfered with his occupation, hemiplegia suddenly occurred when he was running to overtake an uncle whom he saw walking in front of him. Sometimes the symptoms follow a bronchial catarrh, as was the case with a man, aged forty-seven, who became notably breathless during an influenzal attack, though the evidence of pulmonary disturbance was trivial. Sometimes they arise without obvious cause. A man, aged sixty, who had not been looking well for some months according to his medical attendant, whom, however, he had not consulted, took a hot bath one night at 10 p.m. At midnight, when lying quietly in bed reading, he suddenly became extremely breathless and had to sit up in bed. The dyspnœa passed off in ten minutes, but from that time he was always conscious of his heart's action as a constant throbbing within the chest.

A farmer's wife, aged sixty, who had always been healthy and active, save for scarlatina at the age of twenty, and blood-poisoning from a prick of the finger at forty-four, noticed in the spring of 1909 that her feet were often swollen at night, and that she became short of breath on trivial exertion; but she was able to continue at work notwithstanding her discomforts. In the spring of 1910 the symptoms became exaggerated, and she was forced to take to bed and to seek medical advice. The aortic lesion was progressive, and the symptoms, in consequence, became more serious.

The early symptoms, when the onset is insidious, are also

often associated with physical exertion. A man, aged forty-six, for instance, who was accustomed to hard physical exercise, admitted that he had been getting more short-winded than he used to be for a year before the symptoms really troubled him.

The onset of symptoms may be due to various causes. In MacDonald's series 5 patients (8.6 per cent.) ascribed their illness to exposure and chill; 5 were admitted suffering from acute rheumatism; 2 were suffering from notable distress in breathing, which had ensued suddenly when they were at work; 3 were admitted unconscious, 1 after an epileptic fit, and 1 hemiplegic. The modes of onset contrast with those of mitral disease, a fact which is to be related to the difference in the incidence of their various causes. In aortic cases these causes are as a rule progressive in their nature, while from the proximity of the coronary arteries to the valve these vessels are extremely apt to be abnormal, and the nutrition of the cardiac muscle suffers in consequence. A rheumatic scar on a mitral valve may cease to contract, and rarely affects the coronary arteries.

The Actual Symptoms of Mitral and Aortic Disease.

—MacDonald investigated the actual incidence of the symptoms in a series of 102 cases of mitral disease and 58 cases of aortic disease, who were admitted into hospital on account of serious symptoms. The figures in the succeeding paragraphs refer to his series.

Dyspnœa.—*The symptoms of mitral disease* are most often respiratory, and 91 of MacDonald's series (89.1 per cent.) complained more or less of breathlessness. In mitral disease the pulmonary circulation is always congested, and the normal respiratory activity is impeded; mitral patients are often cyanosed. Bronchitis is a frequent complication, and occurred in 39 cases (38.2 per cent.). It may, as already mentioned, originate symptoms, and in their presence always intensifies the distress. One of our chronic hospital patients with mitral disease and auricular fibrillation was quite comfortable and able to do light work so long as his chest was clear, but had serious cardiac failure whenever he contracted a cold in the chest.

Pulmonary infarct and hæmoptysis are common in mitral disease, and occurred in 18 cases (17·6 per cent.). Twenty patients (19·6 per cent.) were orthopnœic; in 10 the breathing was difficult; in 3 the dyspnœa was spasmodic and paroxysmal. Orthopnœa is generally accompanied by abdominal distension (due to flatulence or ascites) or by an enlarged liver. The upright position relieves the pressure upon the diaphragm and permits greater excursion. Dyspnœa is frequently associated with auricular fibrillation, which was present in 19 cases (18·6 per cent.). It may of course be due to other causes, but the sterno-mastoid muscles are almost always habitually in action whenever the pulse is permanently irregular.

In *aortic disease* shortness of breath is also the most common symptom, and was present in 54 cases (92·8 per cent.) of MacDonald's series. It is not, however, related so closely to passive venous congestion of the lungs as in mitral disease, for this is minimal or absent unless the ventricle dilates and the mitral valve becomes incompetent. Pulmonary embolism is unusual, and did not occur in this series, so that death seems to occur before any *considerable* strain is thrown upon the right heart. Shortness of breath is frequently due to bronchitis, which was present in greater or less degree in 37 cases (63·6 per cent.) and may also result from systemic ischæmia, being then experienced on exertion. The result is more likely to follow if, as is often the case, the patient is anæmic. The degree of anæmia is rarely considerable, but in one case the red cells only numbered 2,000,000, with a hæmoglobin value of 35 per cent. Auricular fibrillation is uncommon, and was only present in 2 cases (3·4 per cent.).

In aortic cases shortness of breath is not unusual when the patient is at rest (20 cases, 34·4 per cent.), and is often worse at night. Nine patients (15·4 per cent.) were orthopnœic, and 11 had difficulty in breathing; in 6 of these the dyspnœa was spasmodic and paroxysmal. The majority of the patients were over forty years of age (over forty, 33; under forty, 25), and the probability of a toxic origin is, in consequence, considerable. The question is difficult to answer, but 10 of the 13 cases examined *post-mortem* showed some form of nephritis; and of the 6 patients in whom the dyspnœa was

paroxysmal 2 had granular kidneys, while all the others had persistent, though slight, albuminuria.

Cardiac Discomfort.—Forty-nine (48.0 per cent.) of the *mitral* patients complained of palpitation, and 30 (29.4 per cent.) suffered from thoracic pain. In one case typical attacks of angina occurred. The distinction between discomfort and actual pain is sometimes difficult to establish, for the descriptions of the patients vary according to their sensitiveness, and are often evidently inaccurate. A woman, aged twenty-seven, who was suffering from a double mitral lesion, suddenly became very short of breath one morning. The respirations were more frequent than they had been, and the pulse had doubled in rate and was running about 160. At intervals paroxysms of intense dyspnoea ensued, during which her distress became intensified; the attacks lasted for about fifteen minutes. She said that she had no actual pain, but that her discomfort was unbearable, though she had described her previous attacks as “painful.” Paroxysmal tachycardia, then, occasions discomfort, and so may auricular fibrillation and extrasystoles. Pericarditis is probably a more common cause of discomfort than is realized, for local pericarditis, which had given rise to no evident signs during life, is not uncommonly found on *post-mortem* examination. A girl who was suffering from an acute endocarditis complained of pain in the upper part of her chest for a few hours on December 29th. On February 11th the pain recurred during the night, again lasting for a short time. On March 3rd she had severe pain over the heart for twenty-four hours, and on March 5th pericardial friction was audible. It seemed probable that pericarditis was the cause of the recurring attacks, but that the inflammation was local, and subsided on the two first occasions. In another patient thoracic pain was due to arthritis of the sterno-clavicular joints, apparently of rheumatic origin and associated with large crops of subcutaneous nodules.

Epigastric pain was present in 15 cases (14.7 per cent.). In many more the liver was tender on palpation.

Nineteen (32.6 per cent.) of the *aortic* cases complained of palpitation, and 22 (37.8 per cent.) of thoracic pain, which in

17 cases (29·2 per cent.) was præcordial in site. One patient suffered from typical anginous attacks, and 4 experienced a sense of constriction around the chest. Thoracic discomfort is thus almost equally common in mitral and aortic disease; but palpitation is less, and pain is more, frequent in the aortic group. The frequency with which auricular fibrillation occurs in mitral disease (18·6 per cent. mitral cases; 3·4 per cent. aortic cases) explains the more common complaint of palpitation in this group, while the predominance of pain in the aortic cases must be ascribed to the frequency of associated arterial disease involving the aorta and the coronary arteries. Aneurysm occurred in 9 of the aortic and in 2 of the mitral cases. *Epigastric pain*, on the other hand, was unusual, and only present in 2 instances.

Œdema was present in 51 (49·9 per cent.) of MacDonald's *mitral* cases, and was considerable, and invaded the trunk in 22. In some it was trivial and subsided rapidly as soon as the patient was confined to bed, but in others it was extreme, and in 1 case the skin of the legs gave way and the fluid drained through the apertures. The four causes of œdema in heart disease, cardiac failure, renal disease, anæmia, and obstruction of veins, may all be active in a cardiac case. The first three are often present; the last is unusual. One of our patients, however, complained of pain in her right calf, which was tender to palpation; the overlying skin became red and glazed. The discomfort subsided in a couple of days, but a fortnight later the left leg became œdematous, and the femoral vein tender and cord-like. A pulmonary infarct occurred, but she made a good recovery. Renal œdema is universal, while cardiac œdema is distributed in accordance with the law of gravity. The scalp frequently pits on pressure in renal disease, but we have never found this to occur in cases of cardiac failure where the kidneys seemed to be normal. Local œdema owns a local cause.

In *aortic* disease œdema is slightly less frequent, and is rarely considerable. It was present in 24 patients (41·2 per cent.), but was only considerable in 5.

In exceptional cases, however, it may be both persistent and extreme, and in one old lady, aged sixty, who was

admitted into hospital it was present for twenty-one months, for ten of which she was, in consequence, confined to bed. On her admission the œdema was extreme, and practically universal, and involved the serous sacs to such a degree that paracentesis was required. The mitral valve, however, was incompetent as well as the aortic, and the right heart was notably dilated.

Gastric Symptoms are frequently present in *mitral* disease. Twenty-two patients (21·5 per cent.) complained of anorexia, 34 (33·3 per cent.) of vomiting, 12 (11·7 per cent.) of varying gastric discomfort. One patient suffered from acute gastritis.

In *aortic* disease, on the other hand, gastric symptoms are uncommon. Six patients (10·3 per cent.) complained of anorexia, and 4 (6·8 per cent.) of gastro-intestinal discomfort.

Embolic Phenomena are not uncommon in *mitral* disease, and may be multiple and affect many organs. Sometimes infarcts are numerous.

In one case the patient, a married woman, aged forty-six, had suffered for some years from shortness of breath and palpitation on exertion, but was able to do her household duties without distress. Two months before admission she ran a short distance to catch a tram-car, and, on arriving beside it, was so weak that she was unable to get into it. She felt very breathless and faint, but after leaning against a wall for ten minutes was sufficiently recovered to walk, with her son's assistance, the few hundred yards to her house. She had great difficulty in ascending the stairs, and had to take a rest on several occasions. Next day she felt much better, and got up for a few hours, and within a week was doing her usual work. Two weeks later, when alone in her house, she suddenly, without obvious reason, experienced a gripping pain in the abdomen, which was so severe that she fell to the floor, where she lay for five hours, until her husband returned and assisted her to bed. The pain persisted for a day or two, but gradually lessened, though slight attacks recurred from time to time. The palpitation became more troublesome, and continued even when she was at rest. On the day before admission, when she was sitting in a chair, she suddenly felt extremely weak, and vomited, but the discomfort ceased within ten minutes. On admission to hospital, on February 15th, she was seen to be fairly well developed but poorly nourished, and white hair made her look older than her years. She lay easily in bed, and there was no œdema, but the liver was enlarged and tender, rhonchi were audible all over the chest, and the pulse was frequent and extremely irregular (auricular fibrillation). Both sides of the heart were enlarged. No murmurs were audible, but the first sound at the apex

was short and sharp, with an abrupt commencement, and the second pulmonic sound was emphatic. The urine contained blood and albumin in amount, and numerous tube-casts. Some improvement occurred within the next few days, but on the evening of February 28th she experienced pain in her right leg, which prevented her from sleeping; it had disappeared next day, and nothing abnormal could be seen. On March 19th she complained bitterly of numbness in the left leg and foot, but again examination was negative. On the evening of April 6th she suddenly fell back unconscious, her breathing becoming stertorous, and a left facial palsy was apparent. In a few hours consciousness returned. She had a restless night, but next morning spoke clearly and intelligibly; a slight paresis was evident on the left side, but she could move her limbs. On April 15th she suddenly lost consciousness for ten minutes, but rapidly recovered. On April 20th she complained of pain in the right side of the abdomen, and felt sick and vomited. Cyanosis became extreme, but the symptoms subsided in a couple of days. The pain and vomiting recurred on May 14th, and lasted again for two days; but on May 18th she suddenly lost consciousness and passed into coma, dying a few hours later.

On *post-mortem* examination many infarcts were found, a small one in the lower lobe of the right lung, four in the spleen, and five in the kidneys. In the brain there was a white cicatricial area above and behind the posterior end of the right internal capsule, and a hæmorrhagic infarct of more recent origin in the pons; the right middle cerebral artery was occluded by an embolus near its origin; the cerebral vessels were not diseased. The mitral valve was the site of an extreme button-hole stenosis; the left auricle was greatly enlarged and its walls hypertrophied. The auricular appendix was particularly large, and completely filled by an enormous tough thrombus, which measured some 3 inches in length, and projected as a polypoid mass into the cavity of the auricle. The free surface was in parts coated with soft recent clot.

The cause of the various symptoms was thus apparent. The abdominal pain was due to splenic infarcts, the pain on the right side to the pulmonary infarct, and the hæmaturia, in part at any rate, to the renal ones. The cerebral symptoms owned a similar cause.

Embolic phenomena are unusual in *aortic* disease. None occurred in this series.

Cerebral Symptoms are more common in *aortic* than in *mitral* disease. Of the *mitral* patients 4 (3·9 per cent.) suffered from hemiplegia, 18 (17·6 per cent.) complained of headache, 9 (8·8 per cent.) of giddiness, 8 (7·0 per cent.) of faintness, and 1 (0·9 per cent.) of noises in the head. In the *aortic* group, 2 (3·4 per cent.) suffered from hemiplegia, 7 (12·4 per cent.) complained of headache, 9 (15·4 per cent.) of giddiness,

3 (5·1 per cent.) of faintness, 6 (10·2 per cent.) of insomnia, and 2 (3·4 per cent.) of a sensation of impending death.

The exact cause of these symptoms may be difficult to determine during life. Two of the mitral hemiplegias were due to embolism, and one to hæmorrhage. One of the aortic hemiplegias had a cerebral hæmorrhage. No *post-mortem* examination was made in the other cases. The headache sometimes suggests a definite cerebral lesion, but is most often associated with high blood-pressure and other evidences of renal disease; sometimes it seems to be due to anæmia. Giddiness is most frequent in patients with degenerate arteries, while faintness is not necessarily associated with anæmia or extreme cardiac weakness. In one of our patients who fainted frequently, as a rule after exertion, the only other sign of cardiac disability was very slight œdema of the shins. In some patients fainting is due to flatulence causing reflex vagal inhibition of the heart; in others "faints" are probably *petit mal*.

Of **general symptoms** the most frequent is weakness, which was present in 33 *mitral* cases (32·3 per cent.) and in 13 *aortic* (22·4 per cent.) patients. Three mitral patients complained of coldness of the extremities and one of epistaxis. One aortic patient made complaint of failing eyesight.

CHAPTER XVIII

THE DIAGNOSIS OF CHRONIC VALVULAR DISEASE

THE diagnosis of the various valvular affections is made on examination of the patient. It is impossible in this place to consider all the details of physical examination, but some points require a little special consideration.

The Significance of Physical Signs.—Only a portion of the heart is directly related to the front of the chest, as the lungs overlap all but a small part of the right ventricle (Fig. 197). The apex impulse is thus felt through a considerable thickness of pulmonary tissue, and its character may be modified by variations in the condition of the lungs or pleuræ, quite apart from any alterations in the heart itself. It is, for example, temporarily weakened on full inspiration and strengthened on full expiration, and it may be permanently abolished by emphysema, or its area may be increased by retraction of the lungs. In healthy individuals it is freely movable, and may be felt a couple of inches outside its usual site when the individual lies on his left side. The *absence* of these physiological variations is important, as it shows that the anterior margin of the left lung is fixed, or that the apex of the heart is adherent to the anterior chest wall.

Pulsation on the front of the Chest.—In most healthy persons the apex impulse is the only appreciable *pulsation* on the front of the chest. It is a definite forward thrust or protrusion, which lasts for about a third of a second, and precedes the appearance of the carotid pulse. It is produced by the cardiac systole, which alters the shape of the heart as well as its relation to the chest wall, the apex being tilted forward and to the right, and the long axis of the heart being slightly lengthened; coincidently the transverse diameter of the

heart is diminished. If the interspaces are wide and the chest wall is thin, a recession may be observed over the right ventricle between the apex and the sternum.

It is often difficult to distinguish, either by the eye or by the hand, between a protrusion and a recession, for in both a

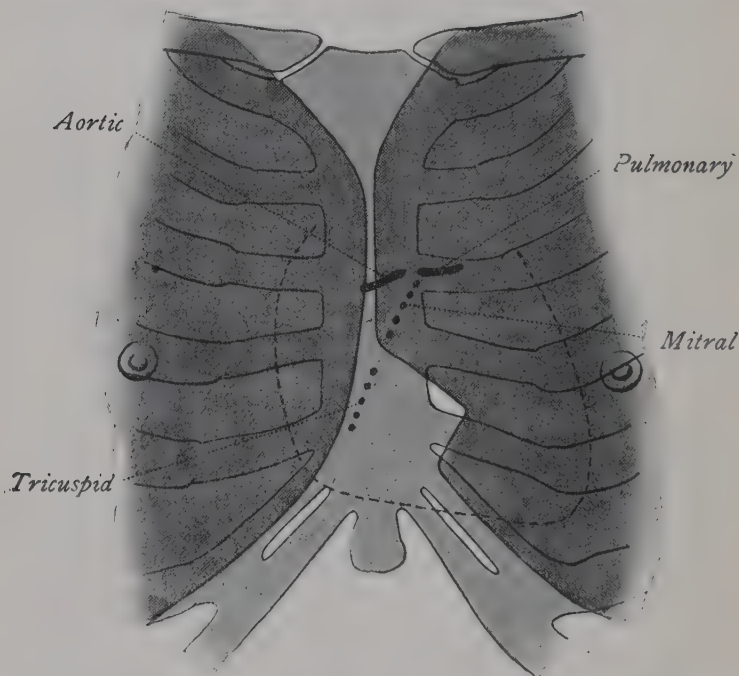


FIG. 197.—DIAGRAM SHOWING THE SURFACE RELATIONS OF LUNGS, HEART AND CARDIAC VALVES (A. KEITH).

forward movement occurs, though at different periods of the cardiac cycle. With graphic methods the distinction is easy, as the arterial pulse can be taken as a guide.¹ Normally

¹ Apart from graphic methods the recognition of the different phases of the cardiac cycle is most readily appreciated by watching the apex impulse and palpating the carotid pulse. The rhythm of pulsations may also be accurately gauged by watching the pulsations of "flags" of adhesive paper placed over them. A binaural stethoscope with two bells is useful for timing murmurs, one bell being placed where the second sound is distinct, and the other at the point of maximum intensity of the murmur. The coincidence, or otherwise, of the two is readily appreciated.

the apex impulse makes its appearance about 0.10 second before the carotid, and 0.20 second before the radial pulse.

If the left ventricle is hypertrophied, the impulse is wide-spread and well sustained, and its relationship to the arterial pulse is unchanged. If the right ventricle, however, is hypertrophied, the apex proper may be displaced backwards from the chest wall, the impulse which is felt being due to the con-

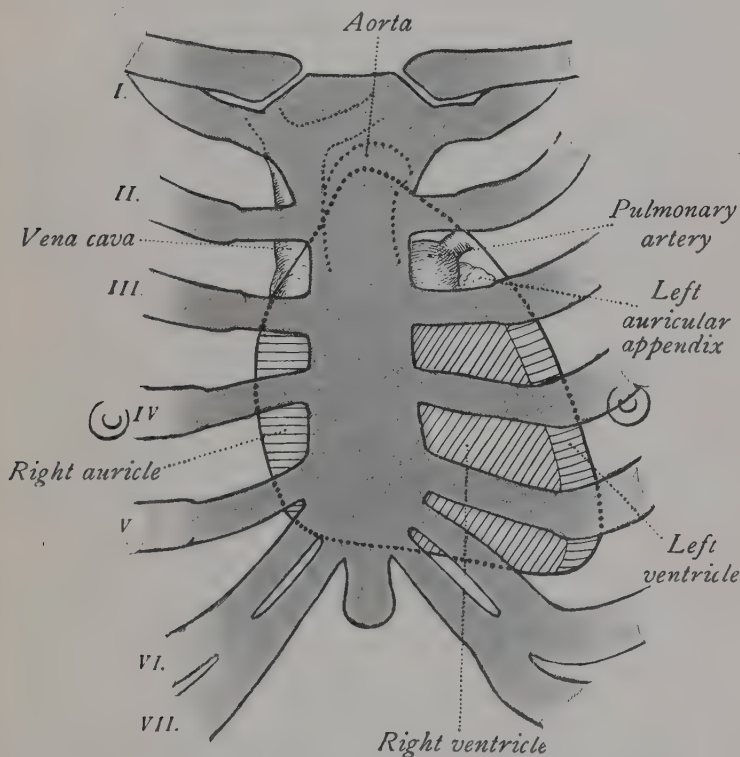


FIG. 198.—DIAGRAM SHOWING THE SURFACE RELATIONS OF THE PERICARDIUM, HEART, AND GREAT VESSELS (A. KEITH).

traction of the underlying right ventricle. The rhythm of the impulse is thus altered, for during systole the transverse diameter of the heart is lessened and a recession occurs, the cardiogram being "inverted" (Fig. 199).

In pathological conditions pulsations may be present in various parts of the chest, and their nature can only be recog-

nized by appreciation of their character and rhythm. Epigastric pulsation, for example, may be due to the pulsation

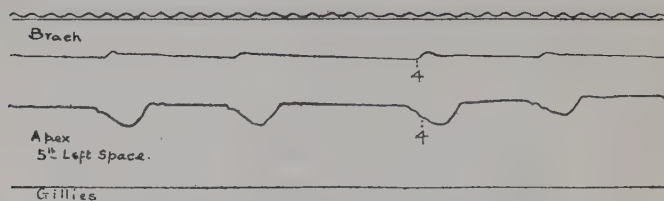


FIG. 199.—“INVERTED” APEX IMPULSE.

of the abdominal aorta (Fig. 200), to true hepatic pulsation from tricuspid regurgitation (Fig. 201), or to communicated

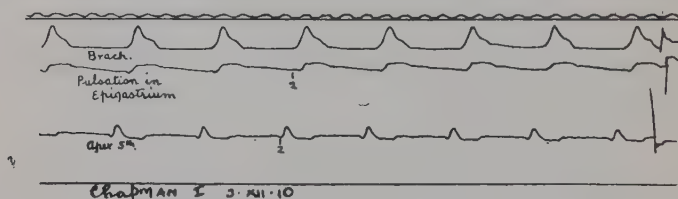


FIG. 200.—EPIGASTRIC PULSATION SUCCEEDING THE APEX IMPULSE: ABDOMINAL AORTA.

pulsation from the heart itself (Fig. 202). The first succeeds the apex impulse, and is practically synchronous with the

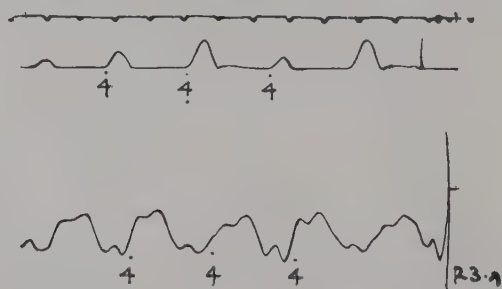


FIG. 201.—EPIGASTRIC PULSATION (LOWER TRACING) PRECEDING THE BRACHIAL PULSE (UPPER TRACING), AND SYNCHRONOUS WITH THE APEX IMPULSE AND POSITIVE, DUE TO HEPATIC PULSATION FROM TRICUSPID INCOMPETENCE.

brachial pulse, and the others are synchronous with the apex impulse and precede the brachial pulse; but if the pulsation is due to the contraction of the right ventricle (Fig. 203) the

tracing is inverted. The pulsation so often seen in the second left interspace close to the sternum is generally due to the pulsation of the pulmonary artery, as the main wave succeeds

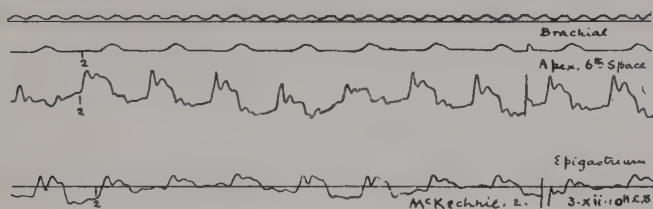


FIG. 202.—EPIGASTRIC PULSATION SYNCHRONOUS WITH APEX IMPULSE AND POSITIVE: LEFT VENTRICLE.

the apex impulse and precedes the brachial pulse (Fig. 204). But it may be synchronous with the apex, and either a protrusion or a recession, the first if it is due to the conus arteriosus,

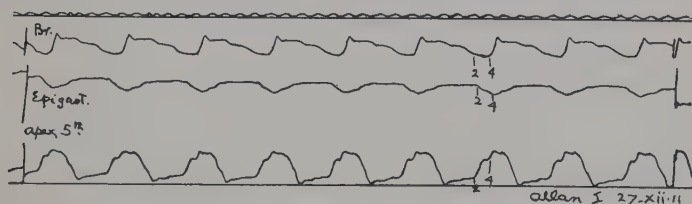


FIG. 203.—EPIGASTRIC PULSATION SYNCHRONOUS WITH APEX IMPULSE, BUT INVERTED: RIGHT VENTRICLE.

the second if it is due to contraction of the right ventricle. The contraction of the auricle may be represented upon this tracing, usually as a positive wave, more rarely inverted.

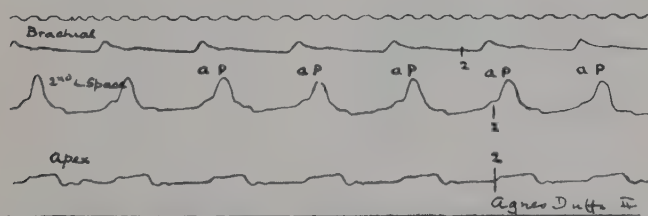


FIG. 204.—CURVES OF BRACHIAL PULSE, PULSATION IN SECOND LEFT INTERCOSTAL SPACE AND APEX IMPULSE.

Pulsation to the right of the sternum generally shows an inverted wave, as it is most frequently due to the contraction

of a dilated right heart (Fig. 205). But the wave may be positive if it results from the reflux of blood through an incompetent tricuspid valve.

Pulsation on the front of the chest may be produced in

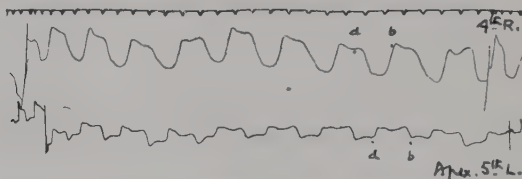


FIG. 205.—PULSATIONS FROM FOURTH RIGHT INTERCOSTAL SPACE AND APEX IMPULSE.

The pulsation on the right side is inverted.

many ways, for the heart is often displaced in pulmonary and abdominal disease, the viscera may be transposed or an aneurysm may be present, and the nature of the pulsation can only be recognized by careful examination.

The Area of Cardiac Dullness is often altered by pulmonary disease. Under normal circumstances it is an

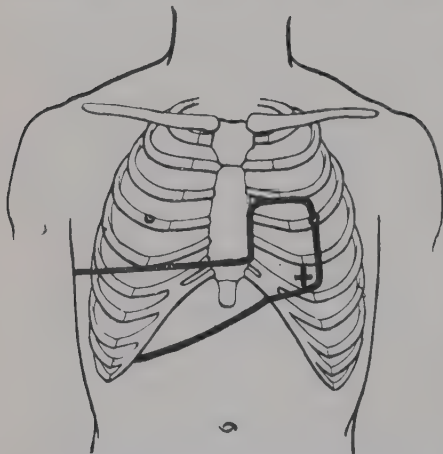


FIG. 206.—DIAGRAM SHOWING AREA OF DULLNESS OF HEART AND LIVER.

inaccurate guide to the size of the heart, for, though the left and the upper margins can be delimited fairly accurately, the right margin is always within the right border of the heart, as the sternal note is representative of the structures underlying it as a whole, and the lower margin runs into the area of hepatic dullness. The so-called areas of superficial and deep dullness are notoriously

inaccurate. A medium strength of stroke yields the best result, and the physician must be careful to use the same

technique habitually and to form *his own estimate* of the *normal dimensions* of the area (Fig. 206).

The cardiac dullness in healthy individuals varies appreciably with variations in respiration, being diminished during full inspiration and *vice versa*. It may be notably lessened in emphysema and apparently increased in pulmonary consolidation or retraction. *Absence* of the physiological variations is an important sign, for it indicates fixation of the anterior margins of the lungs.

The Cardiac Sounds.—The *character* and the *area of audibility* of the cardiac sounds and valvular murmurs require careful examination. The first sound is due to the contraction of the ventricular muscle and to the closure of the auriculo-ventricular valves. If the ventricle is powerful, the sound is long and dull, as the muscular element is well marked; but if the muscle is weak, the valvular element becomes predominant, and the sound becomes *shorter* and *sharper*, and approximates in character to that of the normal second sound. The second sound is due to the closure of the arterial valves. It is loud if the arterial pressure is high, and weak if the pressure is low, or the valves are stiff and immobile; absence of the second sound always indicates a gross valvular defect. But the character of the sounds may be greatly altered by the thickness of the overlying structures, and they are often weakened in fat and in very muscular individuals, and in emphysematous patients; and they may be loud in thin people or in those whose lungs are retracted from the front of the chest, quite apart from any real alteration in the heart itself. The loudness of valvular murmurs may, of course, be affected in the same way. An intoned or ringing second sound indicates stiffness and enlargement of the aorta, rather than changes in the aortic cusps.

The *conduction* of sounds and murmurs is due to several factors: (1) The site of production of the sound. The second sound is loudest at the base, and the first sound over the ventricles. (2) The direction of the flow of blood at the time when the murmur is being produced. A systolic aortic murmur is well conducted into the carotid artery, and a diastolic aortic murmur downwards towards the left ventricle.

(3) The relationship of the chambers with which they are concerned to the surface of the chest. The left auricle, for example, is deeply situated, while the left ventricle is relatively superficial. In mitral stenosis the presystolic murmur is heard best at the apex, because this is the nearest accessible spot to the mitral valve, and the current of blood which produces the abnormal sound is flowing from auricle to ventricle.

In mitral regurgitation, too, the murmur is heard best at the apex of the ventricle, though it must be louder in the left auricle, as the current of blood is flowing from ventricle to auricle; but the distance of the left auricle from the front of the chest prevents it being heard best over the auricle. The murmur of mitral stenosis is rarely heard well at the back of the chest, as the current is flowing from behind forwards; and the murmur of mitral regurgitation is often heard well at the back, near the angle of the left scapula, if the left auricle is notably enlarged, as the current which produces the murmur is flowing towards the back of the chest.

An abnormal conduction of sounds or murmurs indicates some abnormality which occasions it. In consolidations of the middle lobe of the right lung the cardiac sounds are well conducted towards the right nipple. In aneurysm they are well conducted to any region where the aneurysm comes near the surface of the chest. The conduction of the cardiac sounds is thus dependent upon the condition of the structures surrounding the heart almost as much as upon alterations in the heart itself.

The Causes of Murmurs (Fig. 207).—The localization and rhythm of a murmur indicate the site of the valvular lesion, but the character of the murmur conveys little information as to the *grade of the defect*. Its loudness depends upon two factors, the nature of the lesion and the rate of flow of the current of blood; and a murmur may weaken or even disappear in cardiac failure from variations in the second factor, without any change occurring in the valve itself. Other things being equal, a loud murmur is more satisfactory than a weak murmur, for it is indicative of a powerful cardiac muscle.

The duration of a murmur, on the other hand, conveys

definite information of value. A mitral murmur which obscures the first sound and occupies the whole of the short pause indicates regurgitation during the whole of ventricular

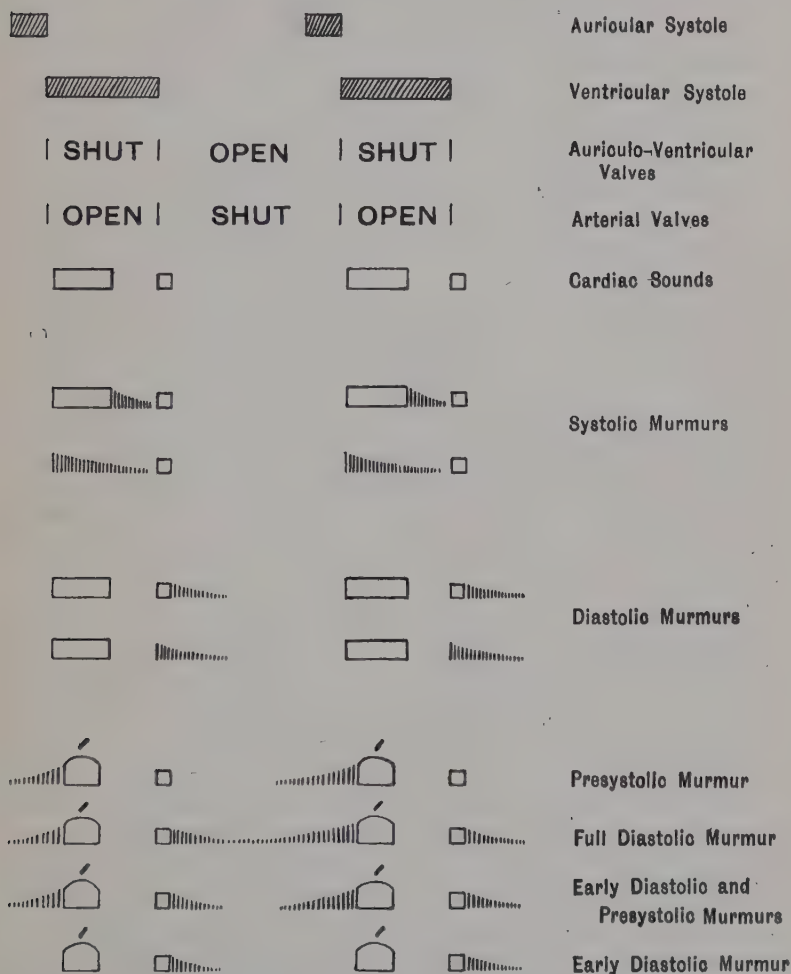


FIG. 207.—DIAGRAM SHOWING THE TIME RELATIONS OF THE CARDIAC CONTRACTIONS, THE CARDIAC SOUNDS AND VALVULAR MURMURS, AND THE CORRESPONDING CONDITION OF THE CARDIAC VALVES.

systole, while a mere whiff succeeding a distinct first sound shows that regurgitation is only occurring during a part of systole.

The character of a murmur may alter from several causes. In acute endocarditis, and less frequently in chronic endocarditis, the valve itself may undergo notable changes. A cusp may be perforated and become notably incompetent, or vegetations may become so luxuriant that a previously existing incompetence ceases. The muscle may become too weak to produce audible vibrations, or with increasing strength may generate a longer and a louder sound. The cardiac rhythm may change. This latter factor has already been considered in some detail (*see* p. 285). In auricular fibrillation the co-ordinate contraction of the auricular muscle ceases, and any murmur produced by auricular contraction ceases too, so that in mitral stenosis, with the onset of fibrillation, the presystolic murmur disappears. Regurgitation in mitral incompetence can be only minimal if the auricle and ventricle contract simultaneously, so that in heart-block, partial or complete, and in extrasystoles, the duration and loudness of a murmur may vary with successive beats; while in nodal rhythm, whether continuous or paroxysmal, a systolic mitral murmur may be weakened constantly, or may disappear altogether.

“Functional Murmurs.”—The presence of a murmur does not necessarily indicate structural disease of the valve. The arterial valves are simple mechanical contrivances which probably act independently of muscular action (*see* p. 295); but the auriculo-ventricular orifices are in large measure closed by contraction of their muscular sphincter, and the valves may become incompetent from muscular weakness without any abnormality of the cusps. Mitral and tricuspid incompetence may thus be temporary if the muscular weakness is due to removable causes. “Curable” mitral regurgitation can be recognized by the absence of any of the factors which produce valvular disease, and by the presence of conditions which produce weakness of, or throw strain upon, the ventricular muscle. Debility, anæmia, etc., represent the former; physical strain and aortic valvular disease the latter.

Murmurs are not infrequently heard in patients whose cardiac valves are normal. They are always *systolic* in rhythm, but may be of various characters, soft or harsh, loud or weak,

long or short ; and they may be present over any part of the cardiac area. According to Sansom,¹ the most frequent site is over the pulmonary artery and the conus arteriosus in the second and third left interspaces close to the sternum (59 per cent.). They may be heard in the aortic area (11 per cent.), and at the apex (7 per cent.) ; and may be present over the conus arteriosus and right ventricle (11 per cent.) ; and at both the apex and the pulmonary area (9 per cent.). The most frequent causes are anæmia and post-febrile debility, and they may also occur in excited hearts (*e.g.*, Graves's disease), in women following child-birth, and in any case where the heart is displaced from its usual site by pleural effusions, abdominal distension, etc. They are generally present in extreme lateral curvature of the spine. The apical murmur is probably always a sign of mitral regurgitation. The basal murmurs are probably produced in the aorta or the pulmonary artery, as the case may be, and may well result from kinking of the vessel in cases where the heart is displaced ; but their nature in other cases is unknown, though numerous theories have been suggested.

Their recognition is usually easy. Organic disease of the pulmonary valve is very rare save in congenital heart disease, in which the disturbance of the circulation is usually considerable, and clubbing of the finger-tips, cyanosis, or other congenital defects, often co-exist ; the right heart is generally enlarged in consequence of the excessive strain thrown upon it. Aortic stenosis without regurgitation is unusual, though a systolic murmur is not uncommon in later life, as the result of some stiffening or roughness of the cusps unaccompanied by definite narrowing of the orifice ; in this case the second sound is generally abnormal, and emphatic or intoned or weakened, and the left ventricle is more or less hypertrophied. In consequence the presence of a systolic basal murmur *as an isolated sign* is of little value as evidence of organic disease. Obvious causes of functional murmurs, too, are usually apparent in these cases, and the *bruit de diable* is frequently heard on auscultation over the jugular vein. The size of the heart is rarely increased, and the second sound is of normal char-

¹ Sansom, A. E., *The Diagnosis of Diseases of the Heart*, Lond., 1892, 273.

acter. The murmur is usually not loud, and is badly conducted, and is *variable* or perhaps inconstant, according to the attitude of the patient and the phase of respiration. It is almost always best marked when the patient is recumbent, and less distinct or absent when he assumes the upright position; and it can often be altered by the cessation of respiration, either on full inspiration or full expiration; while it may only be present when the patient is at rest, disappearing on even slight exertion. A similar variability may also occur with the apical systolic murmur. As a rule this murmur observes all the distinctions which connote the presence of mitral regurgitation, though it disappears when the patient's health and the tone of the muscular sphincter are restored. In other instances, however, the murmur itself is suggestive of a functional cause, being most distinct outwith or inside the apex impulse and not over it, and varying with the respiratory phases, waxing during inspiration and waning during expiration. Cessation of respiration on full expiration generally causes this murmur to disappear (cardio-pulmonary murmur).

Too much reliance, however, must not be laid upon the auscultatory signs, and the case must be considered on general lines. The presence at the time, or previously, of any disease such as rheumatism, which is commonly accompanied by endocarditis, always turns the bias in favour of organic disease of the valve.

Musical murmurs are said to be due as a rule to the projection of a "tag" of valve into the blood-stream, and, in consequence, are most common in cases of acute endocarditis.

In the exceptional case mitral incompetence from dilatation of the orifice without structural damage to the cusps may persist indefinitely. This was the case in a collier who died at the age of fifty from cardiac failure. He came under observation for the first time at the age of forty, with auricular fibrillation. The symptoms were then associated with bronchitis, but subsequently occurred after physical over-exertion. On his early admissions into hospital a mitral systolic murmur was audible for a few days, but disappeared when compensation became established. But it was constantly present for the last seven years of his life. The heart, at *post-mortem*,

weighed 37 ounces. The mitral valve was not deformed, and only showed one little plaque of atheroma upon its anterior cusp. The orifice was considerably enlarged. The incompetence must thus have been due to muscular weakness, but notwithstanding he worked for 79 of the last 108 months of his life, first at the face and subsequently at the pit-head. His systolic blood-pressure ran about 120 mm. Hg.

Canter Rhythm. Bruit de Galop.—In certain cases three sounds may be audible on auscultation over the heart. The origin of the third sound varies in different cases.

In some instances the third sound is really a murmur. This is often the case in mitral stenosis, an early diastolic murmur being audible at the apex, succeeding and separate from the second sound (p. 326). The auricles are usually fibrillating. In mitral stenosis, too, a murmur may occur in mid-diastole on account of a partial heart-block (p. 117).

In other cases, however, the third sound suggests a cardiac sound and not a murmur. (1) In slowly beating hearts, as has already been mentioned (p. 65), a third sound may be audible during diastole. Its occurrence has no particular significance. (2) In another form the triple sound is heard best, perhaps only, at the base, the second and third elements being similar in character, though the emphasis may be on either. It is evidently due to asynchronous closure of the arterial valves ("reduplicated" second sound). It is often present in healthy individuals at the end of inspiration. In disease it is most common in mitral stenosis, though it also occurs in other cardiac lesions as well as in pulmonary disease, if, from any cause, the normal balance of the systemic and the pulmonary circulations is disturbed. (3) In the last group the sound is not heard best at the base. As a rule it is most distinct over the ventricles, midway between the apex and the xiphoid cartilage. The character of the first two elements is similar and differs from that of the second sound as heard at the base. It has been called "reduplicated" first sound, canter rhythm, or *bruit de galop*, from its resemblance to the noise of a horse cantering—rat-a-tat, rat-a-tat. It is never heard in healthy individuals, and is most common in patients with a high blood-pressure and an enlarged heart,

The cause of its occurrence has been much disputed, but its rhythm and its site point to the ventricles for its origin, and it is possibly due to asynchronous contraction of the two ventricles, on account of disturbance of the normal balance of the systemic and pulmonary circulations. Its presence is always a sign of evil omen, as it only occurs when the heart muscle is failing.

The Physical Signs of Mitral Disease.—The general appearance of mitral patients is often suggestive, but the facies is characteristic only in patients with advanced disease. The face is puffy and congested, the complexion is dusky and the lips and ears are more or less blue. A malar flush is common, and the malar capillaries may be permanently dilated. The hands and feet are often cold and clammy, and in chronic cases a mild degree of clubbing of the finger-tips may be found. Difficulty in respiration may be evident even when the patient is at rest.

Mitral Stenosis.—The *apex impulse* is punctate and not well-defined, and little, if at all, displaced; it may be impalpable. *Pulsation* is often present in the second left intercostal space close to the sternum, and in the epigastrium. There has been much discussion as to the cause of the former pulsation. George Balfour maintained that it was due to the dilated left auricle enlarging forwards in front of the pulmonary artery and reaching the front of the chest. This undoubtedly does occur, but it is rare (we have only seen one example *post-mortem*), while the pulsation in question is of common occurrence. It is generally due, as William Russell has shown, to enlargement of the pulmonary artery or of the conus arteriosus, the left auricle seldom presenting more than the tip of the appendix on the front of the heart, and it is outside the pulmonary artery and well away from the sternum.

In polygraph tracings (Fig. 204) the chief wave usually succeeds the apex impulse, and is undoubtedly derived from the pulmonary artery, though a smaller wave, of auricular origin, often co-exists. Occasionally the chief wave is synchronous with the apex impulse, and presumably due to the contraction of the conus. The epigastric pulsation is generally

inverted, and therefore due to the contraction of the right ventricle.

In certain cases of mitral stenosis the apex impulse is large and well marked, and may be considerably displaced both outwards and downwards. This may result from the co-existence of mitral regurgitation or disease of the aortic valve, which necessarily entail enlargement of the left ventricle. When it obtains in the absence of mitral regurgitation or aortic disease, it is invariably associated with a high blood-pressure, and arterio-sclerosis or fibroid kidneys. In some of these cases the epigastric pulsation is due to the left ventricle; careful examination of the rhythm of the pulsation will decide the point. The sternum, too, may definitely pulsate.

A *thrill* is frequently present and is very characteristic. It commences during diastole as a soft, purring tremor, and, rapidly increasing in intensity, ends abruptly as the apex impulse reaches the hand. It is usually limited to the immediate vicinity of the apex, but it is sometimes widespread, and we have felt it distinctly beneath the xiphoid cartilage, though *post-mortem* the tricuspid valve was not narrowed. In one case with this distribution, tracings showed that the epigastric pulsation was due to the left ventricle, the chest being long and narrow, and the left ventricle hypertrophied and powerful.

The *area of dullness* is as a rule but little altered. In some cases it is enlarged to the right from dilatation of the right heart, or upwards along the left side of the sternum, even as high as the second costal cartilage, from dilatation of the conus or the pulmonary artery (Figs. 208, 209).

The *murmur* is late diastolic (presystolic) in rhythm, and at first soft and distant; but it rapidly becomes loud and rough (*crescendo*), terminating abruptly with a loud "snap," the shortened representative of the first sound. It is well imitated by vocalizing the symbols *rrrb* or *vōōt*. The exact cause of the murmur has been much disputed. It commences during ventricular diastole and ends synchronously with the apex impulse, and thus occurs during the period of auricular systole. It is one of the loudest and roughest of cardiac murmurs, and it seems impossible that it can be produced by

the relatively weak auricular systole ; but its chief cause is undoubtedly auricular, for it disappears when auricular fibrillation occurs. Other factors, however, aid in its production.

In its typical form the murmur begins early in diastole,



FIG. 208.—RADIOGRAM : MITRAL STENOSIS, DILATATION AND HYPERTROPHY OF RIGHT HEART (J. R. R.).

and so softly that the exact commencement is difficult to determine ; the “snap” with which it ends is synchronous with the apex impulse. The apex impulse, however, is not exactly synchronous with the commencement of ventricular

systole, for a certain period elapses before the muscular contraction so alters the shape and position of the ventricle that the impulse is perceptible outside the chest wall. The early parts of the murmur, therefore, are diastolic in time, while the terminal crescendo portion is synchronous, first of all with auricular systole, and, at the very end, with the commencement of ventricular systole ¹ (see Fig. 207, p. 315).

The mechanical disturbance in mitral stenosis is peculiar to this lesion. The obstruction at the valve leads to engorgement within the left auricle, and its muscle in consequence hypertrophies, but as this is rarely sufficient, the left auricle dilates, the pulmonary circulation becomes engorged, and the right ventricle in consequence hypertrophies. At the conclusion of ventricular systole the left ventricle is relatively empty and the intra-ventricular pressure is at a minimum, while the auricular (left) pressure is relatively high, as the chamber has just been refilled from the right heart. If, from the relative difference in pressure within the two cavities, the flow of blood is rapid, a soft murmur may be produced. In mid-diastole the difference in pressure is less, for the ventricle is now partly filled and the auricle partly emptied, and the flow is less rapid and the murmur becomes more faint and

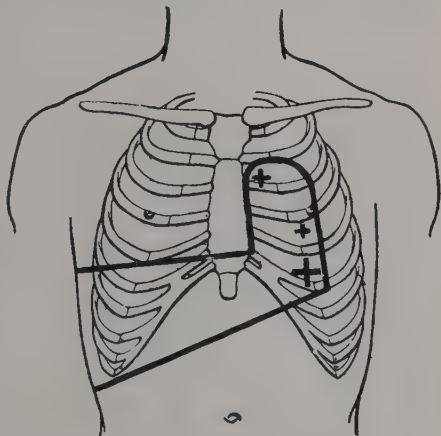


FIG. 209.—DIAGRAM SHOWING AREA OF DULLNESS AND PULSATION (+) IN MITRAL STENOSIS.

This and the following diagrams are actual case records from ward journals.

¹ The time relations of the commencement of ventricular systole and the first sound of the heart have been investigated by means of the phono-electrocardiograph, and it is stated by Fahr* that the first sound begins 0.02-0.03 second after the beginning of the initial ventricular deflexion.

* Fahr, G., *Heart*, 1912-13, iv., 147.

disappears. When auricular systole ensues, the difference in pressure is rapidly augmented, the flow becomes more rapid, and the murmur becomes louder. At the commencement of ventricular systole the intraventricular pressure, on account of the mitral obstruction, is still relatively low, and the auricular pressure is still relatively high, and until the ventricular pressure rises above the auricular pressure as the result of ventricular contraction, the valve will not be closed, and blood will continue to flow from auricle into ventricle. This, of course, only occurs for a very short period, as the intraventricular pressure rises very rapidly ; but, synchronously

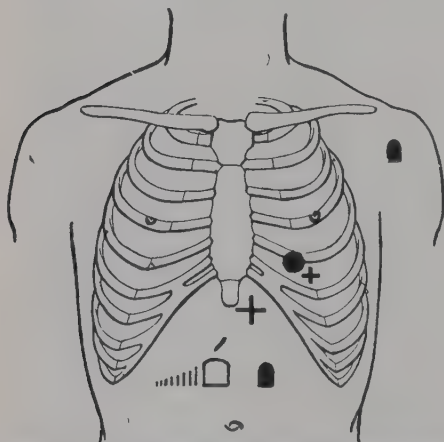


FIG. 210.—DIAGRAM SHOWING DISTRIBUTION OF PRESYSTOLIC MURMUR (●) IN MITRAL STENOSIS.

■ = accentuation of second pulmonic sound.

with the commencement of ventricular systole, the sphincter muscle of the mitral valve and the muscoli papillares contract, so that the shape of the valve and the tension of its cusps are altering while blood is still flowing from auricle into ventricle, and it is just at this moment that the characteristic crescendo part of the murmur occurs. As the current ceases to flow, the “snap” is

heard. The crescendo character, then, is due to blood flowing through the mitral valve at a time *when changes are taking place in the lumen of the orifice and in the tension of the cusps*.¹

The influence of the ventricular contractions in the production of the murmur is shown also by other phenomena. In slight degrees of mitral stenosis the murmur may be absent when the patient is lying down, but becomes distinct on any physical exertion, such as sitting up, an action which has a definite effect on the strength of the ventricular contraction,

¹ Cowan, J., *Glasgow Med. Journ.*, 1898, i., 166.

and but little effect upon the contraction of the auricle; while in cases of partial heart-block, in which the ventricular contractions succeed the auricular contractions by so long a period that auricular systole has ceased before ventricular systole commences, the characteristic crescendo murmur disappears.

The crescendo murmur is usually heard best a little internal to and above the apex impulse, and is limited in its distribution, being generally audible over an area which is little larger than a crown-piece (Fig. 210). The limited area of audibility is proportionate to the area of left ventricle related to the

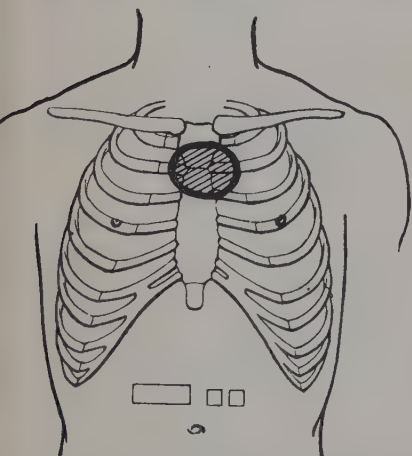


FIG. 211.—DIAGRAM SHOWING AREA OF REDUPLICATED SECOND SOUND.

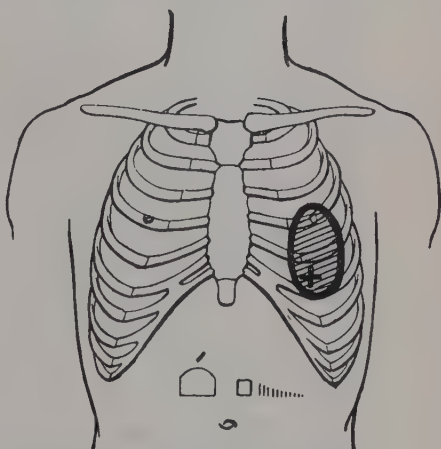


FIG. 212.—DIAGRAM SHOWING AREA OF EARLY DIASTOLIC MITRAL MURMUR.

front of the chest, which, in uncomplicated cases, is small, as the enlarged right ventricle displaces the left backwards and away from the front of the chest; but the area is larger when the left ventricle is enlarged from other causes, and the murmur may then be audible right up to the sternum. The murmur is rapidly lost towards the axilla, though the softer systolic murmur is well conducted in this direction. The reason is obvious. During mitral regurgitation the ventricle is in systole, and the apex is in intimate contact with the ribs, and the vibrations are well conducted along them. In mitral stenosis the murmur is diastolic in rhythm, the apex is not

firmly applied to the chest, and the vibrations are, in consequence, rapidly lost.

If auricular fibrillation ensues, the early diastolic murmur may persist, though the characteristic presystolic murmur

disappears; for when the co-ordinated auricular contraction ceases, the flow of blood at the end of diastole is not sufficiently rapid to produce audible vibrations. The presystolic murmur may also disappear in cases of heart-block, partial or complete, if the normal relationship of the auricular contractions to those of the ventricles is disturbed (*see* p. 117, Fig. 104).

If the ventricle is relatively weak, the early diastolic murmur rapidly succeeds the second sound and quickly dies away (*lubb-te-te*), simulating a doubled second sound, but differing from true doubling in its distribution, the latter being heard best at the base, the former at the apex (Figs. 211, 212). If the ventricle is powerful, the diastolic aspiration may be powerful, and the murmur may be of considerable duration. The murmur is necessarily separated from

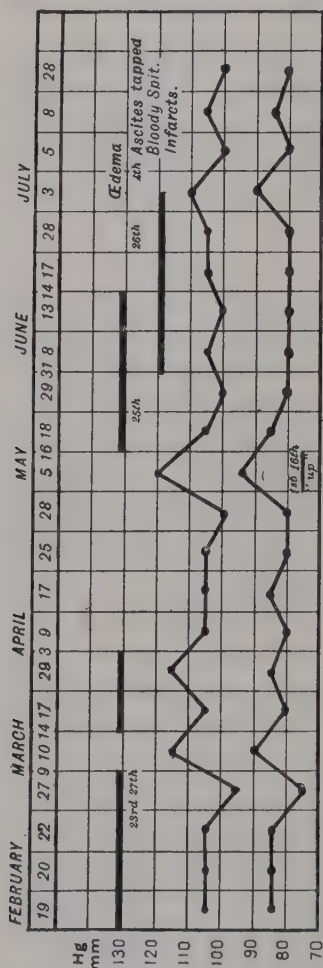


FIG. 213.—CHART SHOWING SYSTOLIC AND DIASTOLIC BLOOD-PRESSURES.
F., aged thirty: Mitral stenosis.

the second sound, for the auriculo-ventricular valve only opens after the aortic valve has closed.

The third murmur of mitral stenosis (Graham Steell's murmur) is of different origin. It is a soft murmur of varying

length, running out of the second sound, and heard best to the left of the sternum in the second or third interspace. It is almost certainly due to relative incompetence of the pulmonary valve, the pulmonary artery having dilated under the stress of the increased pressure within it to such a degree that closure of the valve is impossible.

The *second pulmonic sound* in well-compensated cases is always notably emphatic and the shock of the closure of the valve may be palpable. As compensation fails with dilatation

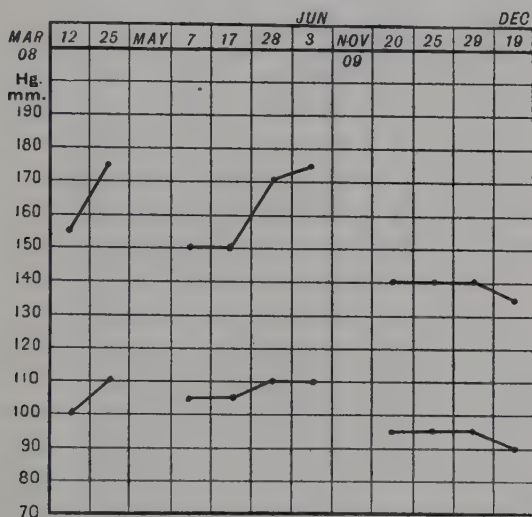


FIG. 214.—BLOOD-PRESSURE CHART.

F., aged forty-six: Mitral disease; cirrhotic kidneys.

of the right heart, it loses emphasis. This, too, as a rule obtains if auricular fibrillation ensues. Very often the two elements of the second sound are not synchronous, and a "reduplication" is audible over the sternum, and may be heard in lessened intensity at the apex.

The *pulse* in mitral stenosis is in no way characteristic. In simple uncomplicated cases it is quite normal so long as compensation is good; but in many cases it is small and hard and well sustained from associated renal disease, a complication which is present in a large proportion of cases, particularly those of some duration (Figs. 213, 214). When

auricular fibrillation ensues, the characteristic irregular pulse makes its appearance, and generally persists until the end. The blood-pressure as a rule falls to some extent when fibrillation occurs.

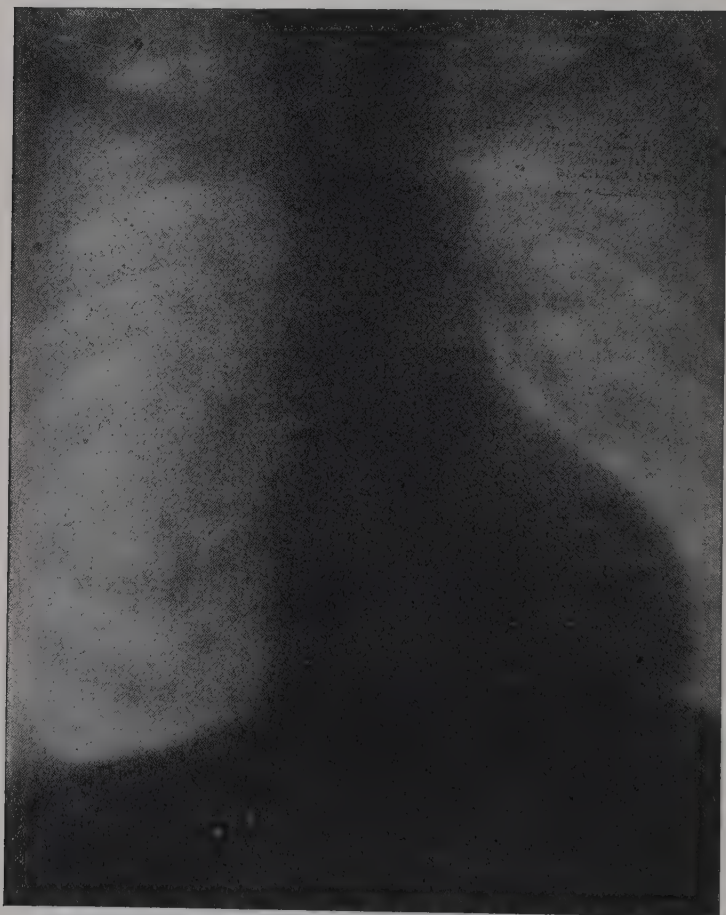


FIG. 215.—RADIOGRAM: HYPERTROPHY OF LEFT VENTRICLE, RENAL CIRRHOSIS (J. R. R.).

In mitral stenosis compensation is at first effected by hypertrophy of the left auricle. In the lesser grades this may be sufficient, but in even average cases it soon fails, and the cavity in consequence dilates, with resultant stasis and

congestion of the pulmonary circuit; to overcome this the right ventricle hypertrophies. It is difficult to appreciate the precise *degree* of the lesion, for the size of the left auricle and of the right ventricle can rarely be measured. Any notable increase in the area of dullness to the right indicates an enlarged right heart (Fig. 216), and the stenosis in these cases is usually considerable; but an enlarged right heart is only present when the right ventricle has dilated under the strain, and compensation is, in consequence, more or less broken. Notable epigastric pulsation and an indistinct apex impulse result from right ventricular hypertrophy; but as they may also occur from other causes, an accurate conclusion is difficult to obtain. If the right ventricle is powerful, the lower end of the sternum may be felt to pulsate; but this depends in great measure on the softness of the ribs, and is seldom well marked save in childhood and adolescence. It must be remembered that mitral stenosis *per se* exerts no influence upon the left ventricle, which, if hypertrophied, is hypertrophied from other causes.

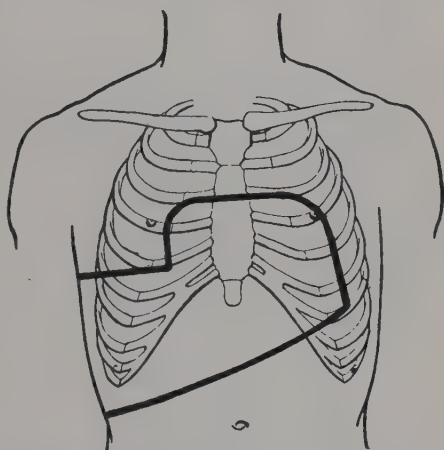


FIG. 216.—DIAGRAM SHOWING AREA OF DULLNESS IN A DILATED HEART.

Mitral Incompetence.—The *apex impulse* is diffuse and heaving, and is displaced to the left and downwards. Pulsation may be felt in several interspaces, and is usually also evident in the epigastrium. A *thrill* is sometimes felt at the apex; it is diminuendo in character, and accompanies and succeeds the apex impulse; it is more often absent than present. The *area of dullness* is increased to the left and downwards in corresponding proportion to the displacement of the apex (Fig. 217). The *murmur* is diminuendo, and

systolic in rhythm, replacing or following the first sound. It is usually soft and blowing, and is less harsh than an aortic systolic murmur. It is heard best at the apex, and is well conducted into the axilla along those ribs with which the apex is in contact (Fig. 218). When the murmur is loud, it may be audible all over the left chest; and it is often distinct at the back, with maximal intensity at the angle of the left scapula, and in a lessened degree along the spine (Fig. 219). The *second pulmonic* sound is emphatic. The second sound

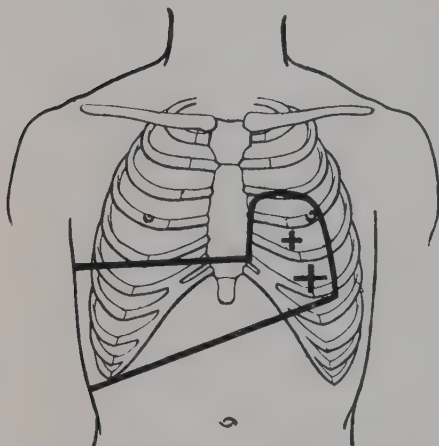


FIG. 217.—DIAGRAM SHOWING AREA OF DULLNESS AND PULSATION IN MITRAL REGURGITATION,

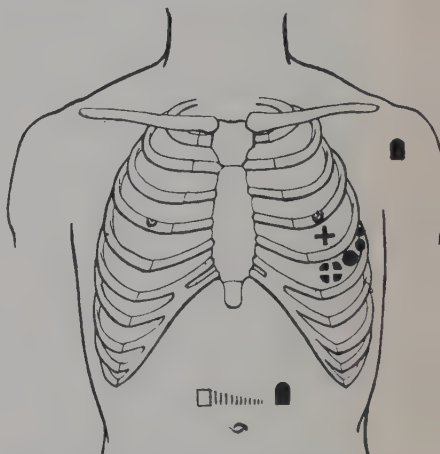


FIG. 218. —DIAGRAM SHOWING DISTRIBUTION OF SYSTOLIC MURMUR IN MITRAL REGURGITATION.

■ = accentuation of second pulmonic sound.

at the base may be doubled, but this is more frequent in stenosis than in incompetence.

The *pulse* is normal in character in simple, well-compensated cases; but is well sustained if, as frequently happens, renal fibrosis co-exists. When auricular fibrillation ensues, the characteristic irregular pulse makes its appearance. The blood-pressure in simple, well-compensated cases is about, or slightly below, the normal reading. In cases where renal fibrosis or arterio-sclerosis co-exist, the pressure is elevated, sometimes to very high readings; it is generally, but not invariably, high in the presence of oedema (*see* p. 507).

The murmur sometimes shows notable variations in its character with successive beats. This is particularly the case in heart-block (*see* p. 121) when the auricular and the ventricular contractions coincide, as regurgitation is prevented or is minimal, and the murmur is, in consequence, absent, or weakened and shortened. A similar phenomenon may occur if auricular fibrillation ensues, for the auricle may then become so distended that free regurgitation is impossible, a result which is most likely to happen if the ventricular contractions are frequent. A similar explanation, too, accounts for the frequent absence of a regurgitant murmur in well-marked cases of mitral stenosis, where *post-mortem* the valve is seen to be so hard and shrunken that the orifice can hardly be closed, even if considerable force is exerted. The obstruction in these cases is often extreme, and the congestion in the auricle too great to allow any appreciable regurgitation from the ventricle during ventricular systole.

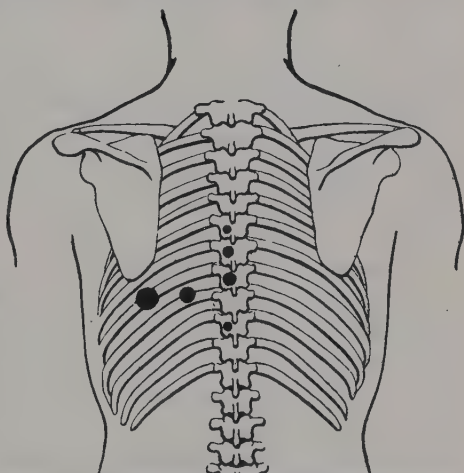


FIG. 219.—DIAGRAM SHOWING DISTRIBUTION OF SYSTOLIC MURMUR OF MITRAL REGURGITATION.

Mitral regurgitation is compensated by alterations in the left auricle and ventricle, and in the right ventricle. The capacity of the left ventricle is necessarily increased, for, if blood flows back through the mitral orifice during ventricular systole, the ventricle must enlarge to accommodate the amount returned into the auricle as well as the normal amount for the aorta; and the wall is hypertrophied on account of the extra work which is entailed. During ventricular systole the left auricle is over-distended, for it receives blood from the left ventricle as well as from the right, and the auricular wall, in consequence, hypertrophies. The right ventricle, too, hyper-

trophies, as its work is increased by the heightened pressure within the pulmonary circuit.

The *degree* of the lesion may be gauged by the degree of enlargement of the left ventricle. The length of the murmur, too, is of value in this respect, as a short murmur occurring after the first sound indicates a short duration of the regurgitation, and in consequence a minimal leak. If the murmur is well heard at the scapular angle, the lesion is probably always organic and gross, for it is only conducted in this direction in cases where the left auricle is dilated and reaches close to the back; and this will not occur in slight or in functional insufficiency. In well-marked regurgitation the second pulmonic sound is notably emphatic. If the right ventricle is greatly hypertrophied, the lower end of the sternum may pulsate, and epigastric pulsation be well marked. The relationship between the size and strength of the pulse and the apex impulse should be contrasted, for in well-marked regurgitation the pulse is small and soft, though the apex impulse is diffuse and powerful.

In cases of mitral disease where compensation has failed, the physical signs alter. The pulsation in the apex region becomes more diffuse and less powerful, and is often better seen than felt, and is not well sustained. Epigastric pulsation is more marked, and pulsation may be felt in the fourth and fifth right intercostal spaces. The area of dullness is increased both to the left and to the right. The murmurs become shorter and less loud, and the second pulmonic sound less emphatic; reduplication generally persists. The pulse becomes more frequent and the wave loses in strength, volume and duration.

The Physical Signs of Aortic Disease.—The general appearance of aortic patients may be characteristic. They are often pale, and may appear careworn or anxious, frightened or in pain; the carotid or the temporal arteries may visibly pulsate, and the head may shake with each beat of the heart. These symptoms, however, must be referred more frequently to associated disease than to the valvular lesion itself; for while anæmia occurs in aortic valvular disease, it only obtains when the compensatory arrangements have broken down

and the general nutrition is, in consequence, defective. Anæmia occurs in syphilis, in arterio-sclerosis, and in chronic renal disease, and its presence in aortic valvular disease is most often due to those causes, and not to the cardiac lesion. Anæmia is by no means constant; some aortic patients are plethoric rather than anæmic; others are cyanosed; in some the blood-counts are normal; though, in a general way, a contrast should be drawn between the cyanosis of the countenance in mitral, and the pallor in aortic disease. A similar distinction holds good with regard to the facial expression. The cause of angina pectoris is sometimes cardiac and sometimes aortic, and the frequency with which disease of the aorta co-exists in aortic valvular disease makes pain and anxiety common accompaniments. Even the throbbing of the arteries is not pathognomonic, for they throb whenever a large quantity of blood is suddenly thrown into relatively empty vessels; and it may be notable in exophthalmic goitre and in excited chlorotic girls, quite apart from any valvular disease.

Aortic Stenosis.—Pure aortic stenosis is comparatively uncommon, and we have seen only two or three specimens. In one case in which the valve presented an appearance very similar to that shown in Fig. 181 a systolic murmur alone was present, so that regurgitation must have been minimal. The patient, who was aged sixty-two, was admitted with œdema, Cheyne-Stokes breathing, etc., and died from progressive cardiac failure. A loud, harsh murmur was audible all over the cardiac area, replacing the first sound and occupying a considerable portion of the short pause. It was loudest at the aortic cartilage, where the second sound was inaudible; a very faint second sound could be heard at the apex. The heart on *post-mortem* examination was found to weigh $24\frac{1}{2}$ ounces. The aortic cusps were adherent to each other for the greater part of their extent, and were thickly infiltrated with large calcareous masses. The lumen was greatly narrowed, and the cusps were practically immobile, and to the water-test notably incompetent, though no diastolic murmur was audible during life.

The *apex impulse* is large and heaving and well sustained,

and is displaced downwards and in a lesser degree to the left. A *thrill*, which is systolic in rhythm and usually rougher than the thrill of mitral regurgitation, may be felt at the base. The *area of cardiac dullness* is increased downwards and to the left in corresponding proportion to the displacement of the apex. The *murmur* is diminuendo, and systolic in rhythm, following or replacing the first sound. It is generally harsh and is often loud, and contrasts with the soft murmur of mitral incompetence. It is heard best at the aortic cartilage, and is well conducted upwards into the neck along the carotid and

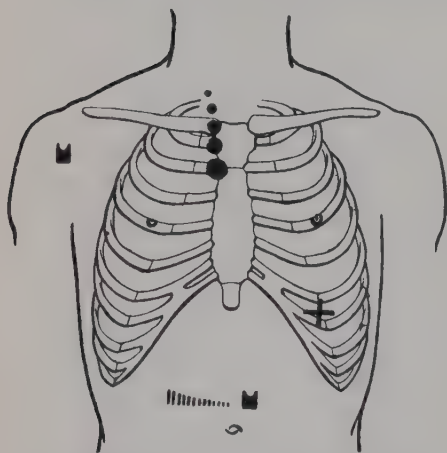


FIG. 220.—DIAGRAM SHOWING DISTRIBUTION OF MURMUR OF AORTIC STENOSIS.

■ =weakening of second aortic sound.

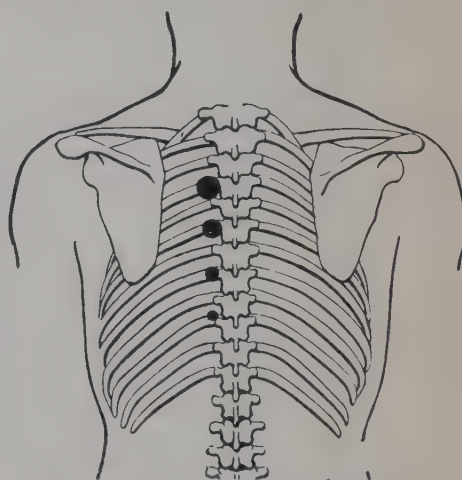


FIG. 221.—DIAGRAM SHOWING DISTRIBUTION OF MURMUR OF AORTIC STENOSIS.

subclavian arteries (Fig. 220). It is sometimes very loud, and may be audible on auscultation over the vertex of the head, the sacrum, and the condyles of the humerus; or even without direct contact with the patient. When heard at the back, it is loudest close to the left border of the vertebral column, about the level of the fourth dorsal vertebra, where the aorta first comes into contact with the spine (Fig. 221). The *second aortic sound* may be weakened or inaudible if the stenosis is considerable and the cusps are stiff. A second sound is, however, generally audible at the aortic cartilage even in well-marked cases, but due to the closure of the pulmonic

valves. The aortic sound alone is heard on auscultation over the carotid artery, and the character of the sound should be gauged by auscultation above the clavicle, so as to avoid this fallacy.

The *pulse* is small, infrequent, and slow, the wave rising gradually beneath the finger. It is well sustained, and the

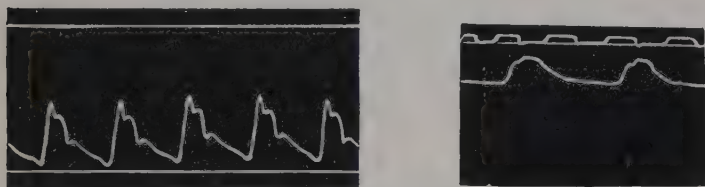


FIG. 222.—SPHYGMOGRAPHIC TRACINGS OF: (1) NORMAL PULSE, AND (2) PULSE IN AORTIC STENOSIS.

artery is well filled between the beats. These characters, however, are probably due to coincident arterial disease rather than to the valvular defect, but the two frequently co-exist. An anacrotic pulse is generally the result of arterial hardening, just as intonation of the second sound is an indication of an enlarged and stiff aorta, rather than an index of the height of the blood-pressure. *Tracings* show the gradual rise of the pulse-wave, the rounded apex, and the gradual fall; the normal waves on the downstroke are usually poorly marked, and the dicrotic wave may be wholly absent (Fig. 222).

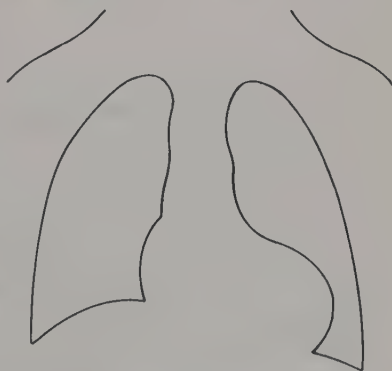


FIG. 223.—ORTHODIAGRAM. AORTIC INCOMPETENCE.

Aortic stenosis is compensated by hypertrophy of the left ventricular muscle, and the degree of the stenosis may be estimated by the degree of enlargement, but here again the condition of the arteries and kidneys must be taken into account, for the hypertrophy may in large measure be due to arterial or renal disease.

Basal systolic murmurs not infrequently obtain from mere roughening or hardening of the cusps with little or no stenosis; the condition can be recognized by the absence of ventricular hypertrophy which accompanies any definite narrowing. The character of the second sound is important, for its absence indicates gross valvular disease.

The relationship between the size and strength of the pulse and apex impulse should also be compared, for in gross stenosis the pulse-wave is small and weak, while the apex impulse is diffuse and well sustained.

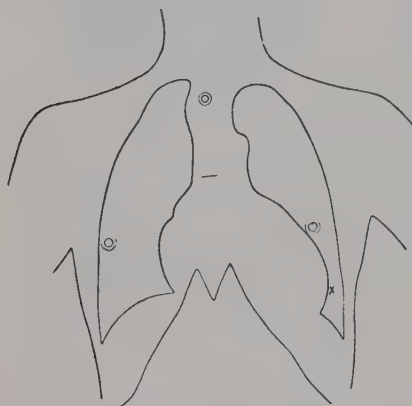


FIG. 224.—ORTHODIAGRAM. AORTIC AND MITRAL INCOMPETENCE. MAN AGED SIXTY-ONE.

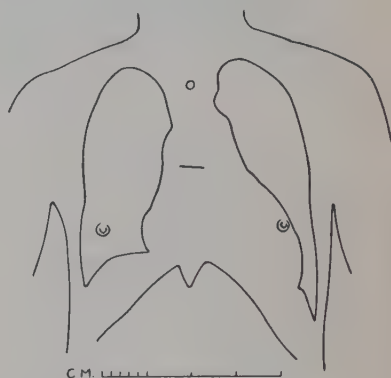


FIG. 225.—ORTHODIAGRAM. AORTIC AND MITRAL INCOMPETENCE.

Aortic Incompetence.—The *apex impulse* is powerful, well sustained and diffuse, and is displaced downwards and to the left. Pulsation may be present in several interspaces. Pulsation, too, is often evident elsewhere; towards the base and in the second and third right interspaces, even in the absence of dilatation of the aorta, and in the carotids and above the sternum. The whole chest may heave. The head is sometimes seen to shake with each systole; and the vehemence of the beats may be so great that they can be felt by the observer seated on the bed without direct contact with the patient. A *thrill* can sometimes be felt over the base, diastolic in rhythm, and succeeding the second sound. It is more often absent than present. The *area of cardiac dullness* is increased to

the left and downwards, in corresponding proportion to the enlargement of the left ventricle (Fig. 226).

The *murmur* is diminuendo, and diastolic in rhythm, succeeding or replacing the second sound, and running for a variable period into the long pause. It is usually soft and blowing, and may be distant and difficult to detect; but it is often loud and superficial; it is sometimes musical in character. It is heard best over the sternum, as a rule with maximal intensity about the middle, opposite to the third or fourth interspaces; and is well conducted down the sternum

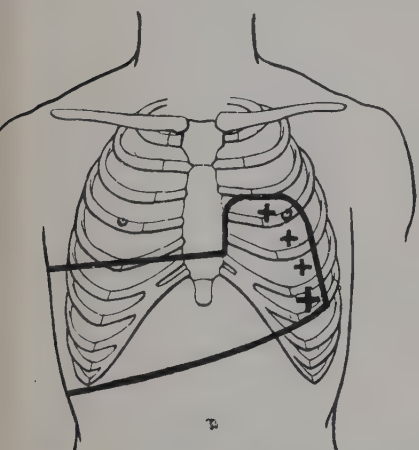


FIG. 226.—DIAGRAM SHOWING AREA OF DULLNESS AND PULSATION IN AORTIC INCOMPETENCE.

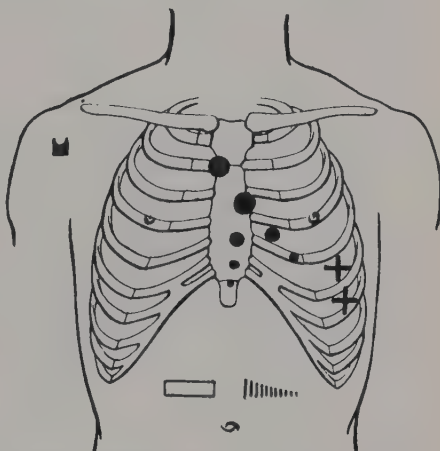


FIG. 227.—DIAGRAM SHOWING DISTRIBUTION OF DIASTOLIC MURMUR IN AORTIC INCOMPETENCE.

■ = weakening of second aortic sound.

to the xiphoid cartilage, and in lessening degree to the apex (Fig. 227). When it is well heard at the apex, it is said (Foster) to indicate a special failure of the left posterior cusp. Sometimes the murmur is heard best at the aortic cartilage (Fig. 228), and it may be conducted into the carotids, but much less frequently than the systolic aortic murmur. Occasionally it is heard best towards the xiphoid end of the sternum. In well-marked cases, with a loud murmur, it may be audible all over the chest.

In the majority of cases the *second aortic sound* persists and precedes the murmur; but in gross defects, when the

cusps are so shrunken or stiffened that they cannot meet, the sound may disappear and be replaced wholly by the murmur.

A presystolic murmur (Austin Flint's murmur) is sometimes heard at the apex in these cases without there being any mitral disease. In the majority of cases, however, in which a presystolic murmur is present, mitral stenosis co-exists.

The pulse, in gross lesions, is large and quick (shotty, collapsing, water-hammer, Corrigan's pulse), rapidly reaching a maximum and rapidly collapsing. This character is exaggerated if the hand is raised above the head, and may be better appreciated if the forearm is grasped by the whole hand than if

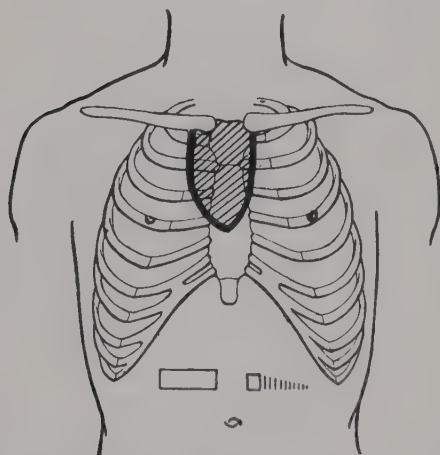


FIG. 228.—DIAGRAM SHOWING AREA OF DIASTOLIC MURMUR OF AORTIC INCOMPETENCE.

the artery alone is felt by the finger. Capillary pulsation may be seen in the finger nails, on the inside of the lips if a glass slide be pressed upon them, or on the brow if the skin is rubbed and rendered hyperæmic. The systolic pressure is high, but the pressure falls rapidly during diastole, and the mean pressure is low relative to the systolic. The *pulse pressure*, the difference between the systolic

and the diastolic pressures, may be great. Its degree is a useful guide to the severity of the leak. In cases of moderate severity it may not exceed the normal variations, 20–40 mm. Hg. A systolic pressure of 150 or 160, with a diastolic pressure of 60 or 70 and consequently a pulse pressure of about 100, is not infrequent in patients with a gross lesion. In one case with a pulse pressure of 105 (systolic, 195; diastolic, 90 mm. Hg.), the left ventricle was very large, the pulse shotty to the finger, and the second aortic sound was absent. The head shook with each systole of the heart. In some instances we have obtained records of a pulse

pressure as large as 150 (systolic, 218; diastolic, 68), or 160 (systolic, 250; diastolic, 90). In the lesser lesions, on the other hand, the systolic and pulse pressures may be within normal limits, such as 120, 40; 120, 35; 115, 20; actual figures from our records. As a rule, however, one or both figures are greater than normal; 155, 55; 150, 60; 135, 50 (*see* p. 297).

When auscultating over the larger arteries, such as the femoral or brachial, a sharp, clear sound synchronous with the arterial pulse may be heard if the valvular lesion is well marked, and particularly if the pulse pressure be very large and there be associated cardiac failure. This systolic sound is seldom heard in any other condition than aortic incompetence, but may be heard if the pulse pressure is unduly large, as in exophthalmic goitre. In aortic incompetence the sound may be so loud as to be audible at a considerable distance

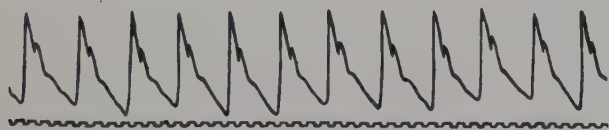


FIG. 229.—SPHYGMOGRAM IN AORTIC INCOMPETENCE.

from the patient. In one of our cases, a man aged twenty-three, the systolic sound produced in the femoral arteries could be heard at a distance of three feet from the patient while the brachial sound could be heard six inches from the arm.

A double murmur of local origin is sometimes audible over the larger arteries, such as the femoral and brachial, on firm pressure with the stethoscope. Pressure over any artery, of course, always causes a single systolic murmur, but the diastolic murmur (Duroziez) probably only occurs in cases of aortic incompetence, though in these it is more often absent than present.

Tracings show an almost vertical abrupt upstroke, a pointed apex, and a sudden fall, the aortic notch being low down and the dicrotic wave poorly developed (Fig. 229).

Aortic regurgitation is compensated by enlargement and hypertrophy of the left ventricle (Fig. 223), and the *degree* of

the regurgitation may be estimated by the degree of the enlargement. Mere hypertrophy of the ventricular wall is insufficient, the ventricular content must be larger; for if the aortic content during diastole is to remain of normal amount, the systolic influx must be abnormally large to compensate the amount lost by regurgitation. In the same way the pulse pressure is an index of the degree of the fault, for the systolic pressure must be abnormally high, if, notwithstanding the rapid fall of pressure during diastole, the mean aortic pressure is to remain normal. The pulse pressure is often a valuable guide in these cases, for it is not related to mitral valvular disease. Not infrequently mitral and aortic regurgitation co-exist, and the enlargement of the left ventricle is due to the two causes combined; and it is difficult to assess the proper value of each individual defect. The point may be determined by the finger, for the pulse is more collapsing and "quicker," the greater the aortic incompetence.

The pulse pressure, however, is only useful as a gauge of the degree of the leak when compensation is effective. Two of our patients had blood-pressures which were similar (115 mm. systolic, and 92 mm. diastolic), but the lesions were in no way comparable. In one there was no evidence of cardiac weakness, the patient being under observation on account of an attack of acute rheumatism, and the lesion was minimal. The other patient was moribund at the time, fainting habitually when at stool, and sometimes when he merely sat up in bed. Muscular weakness was so extreme that the arterial pressure could not be raised to a satisfactory level, the lesion being gross. Coincident stenosis may wholly alter the characteristic pulse of aortic regurgitation if the obstruction is considerable, and the outflow of blood gradual and prolonged.

Weakening or disappearance of the second aortic sound is also an important guide to the grade of the lesion, for the presence of a normal sound indicates that the valves are fulfilling their purpose fairly well, notwithstanding the leak. A well-marked dicrotic wave in the sphygmogram indicates a small leak, and its absence a gross incompetence. The length of the murmur is an indication of the duration of the regurgitation. In slight cases it merely follows the second

sound for a short distance, but in severe leaks may occupy the whole of the diastole.

The Physical Signs of Valvular Disease of the Right Heart.—Valvular disease on the right side of the heart is uncommon. Both the pulmonary and the tricuspid valves may be affected by acute endocarditis, but this is generally malignant in type and secondary to septic foci elsewhere, the organisms being conveyed directly to the heart through the veins; and is rarely of the simple form, so that chronic lesions are unusual. When chronic lesions occur, they are almost always accompanied by disease of the mitral or aortic valves, of older date and more extreme grade. The tricuspid valve is affected much more frequently than the pulmonary.

Congenital defects are fairly common, and affect the pulmonary artery in particular, tricuspid lesions, like those of the mitral valve, being extremely rare. As pulmonary defects arise during the development of the septum of the *truncus arteriosus*, the aortic valve is frequently also involved.

Functional (secondary) incompetence of the tricuspid valve, on the other hand, is of common occurrence. The tricuspid is similar in its structure to the mitral valve, and becomes incompetent if the right ventricle dilates, the muscular sphincter becoming unable to narrow the orifice sufficiently for the cusps to occlude it. The right ventricle is always overworked in mitral disease, and in chronic pulmonary diseases, such as emphysema and fibroid phthisis; and although compensatory hypertrophy ensues and so allows the work to be carried on for a time, it ultimately becomes insufficient, and the ventricle gives way under the strain. In acute pulmonary disease a temporary incompetence is not unusual.

Tricuspid incompetence, even when due to muscular weakness, is always extremely serious, for the strain is thrown upon the right auricle, which is incapable of satisfactory compensatory hypertrophy, so that venous stasis follows. The appearance of œdema in the feet and passive venous congestion of the liver are the result.

Pulmonary incompetence may own a similar cause. In cases of extreme mitral stenosis the blood-pressure in the

pulmonary circuit is extremely high, and the artery may dilate and incompetence ensue without any lesion of the cusps. With improvement the characteristic murmur may disappear. The comparable incompetence of the aortic valve in cases of high blood-pressure is rare, for the aorta is thicker than the pulmonary artery, and its valve remains competent (in experiment) under a pressure which produces notable regurgitation at the pulmonary valve (G. A. Gibson¹).

Tricuspid incompetence is probably the most frequent as well as the most serious valvular lesion, for it occurs in many diseases as a sequel of cardiac failure, and so is the immediate precursor of death.

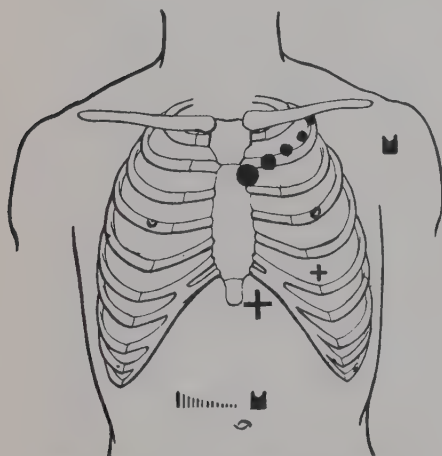


FIG. 230.—DIAGRAM SHOWING THE DISTRIBUTION OF THE SYSTOLIC MURMUR OF PULMONARY STENOSIS.

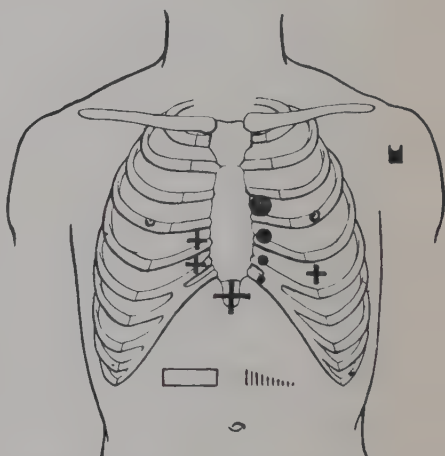


FIG. 231.—DIAGRAM SHOWING DISTRIBUTION OF DIASTOLIC MURMUR OF PULMONARY INCOMPETENCE.

Pulmonary Stenosis.—The *murmur* is diminuendo, and usually loud, harsh and superficial, replacing or following the first sound. It is heard best in the second left interspace close to the sternum, and is well conducted towards the left clavicle (Fig. 230). It is not conducted into the carotid artery. If the murmur is audible at the back the maximum intensity is some distance away from the spine, about the centre of the left scapula. The *second pulmonic sound* is

¹ Gibson, G. A., *Edin. Med. Journ.*, 1880, xxv., 982.

weakened or absent. A *thrill* which is systolic in rhythm is generally present, with maximum intensity in the second left interspace. The *right heart* is hypertrophied, and pulsation may be evident in the epigastrium, at the xiphoid end of the sternum, and in the fourth and fifth right interspaces.

Compensation is effected by hypertrophy of the right ventricle. To gauge the degree of the stenosis is difficult; but the hypertrophy is considerable, and the stenosis is great, if the lower end of the sternum pulsates. The duration of the murmur is an important guide to the grade of the defect. Absence of the second pulmonic sound indicates a gross lesion of the valve.

Pulmonary Incompetence.—The *murmur* is diminuendo, and usually soft and blowing in character, and is diastolic in rhythm, replacing or following the second sound. It is heard best in the second or third left interspace close beside the sternum, and is conducted downwards, and in the direction of the xiphoid cartilage (Fig. 231). The *second pulmonic sound* may be weakened or absent. A soft diastolic *thrill* may be felt to the left of the sternum. The *right heart* is hypertrophied, and pulsation may be present in the epigastrium, at the xiphoid end of the sternum and in the fourth and fifth right interspaces.

The distribution of the murmur, if it is loud, thus closely resembles that of the murmur of aortic regurgitation. The important differences are the enlargement of the *right heart*, the interference with the *pulmonic* second sound, and the *absence* of the characteristic pulse of aortic disease.

Compensation is effected by enlargement and hypertrophy of the right ventricle, but the degree of the lesion is difficult to gauge. If the lower end of the sternum pulsates, the hypertrophy is considerable and the incompetence is great. Absence of the second pulmonic sound indicates a gross lesion of the valve.

Tricuspid Incompetence.—Pulsation is generally evident at the lower end of the sternum and in the epigastrium, and often in the fourth and fifth right interspaces. The *apex impulse* is not displaced, but it may not be palpable. A *thrill* is rarely

present. The *area of dullness* is increased to the right, but does not extend upwards above the third costal cartilage (Fig. 216). The *murmur* is diminuendo, and systolic in rhythm, replacing or succeeding the first sound; it is usually soft and blowing in character, is heard best at the xiphoid end of the sternum, and is well conducted towards the right nipple (Fig. 232). At the back its maximum intensity is at the angle of the right scapula. The *second pulmonic sound*

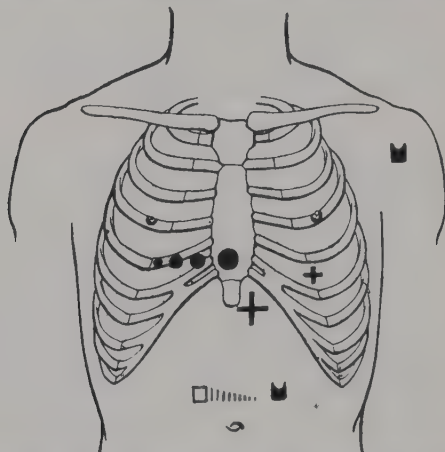


FIG. 232.—DIAGRAM SHOWING DISTRIBUTION OF SYSTOLIC MURMUR OF TRICUSPID INCOMPETENCE.

is weakened. The jugular veins are usually greatly distended, and may pulsate forcibly. The liver may show an expansile pulsation, and generally is notably enlarged and tender on palpation.

Tricuspid incompetence is compensated to some extent by dilatation of the right ventricle and hypertrophy of its wall, and by dilatation and hypertrophy of the right auricle. The results are, however, less satisfactory than in mitral incompetence, for failure of the right auricle entails general venous stasis, as it has no assistance, when in difficulty, that can be compared to the action of the right ventricle in failure of the left auricle. The degree of auricular distension may be measured by the increase in the size of the cardiac dullness to the right, or by the size of the distended veins and the

congested liver and the amount of œdema. If the liver is cirrhotic, however, its enlargement is rarely great. The character of the cervical pulse is of assistance, for in free tricuspid regurgitation the auricle is rapidly distended by the reflux from the ventricle, and the *v* wave occurs prematurely, and is merged into the carotid wave, the *x* depression being lost (see Fig. 45, p. 62).

Tricuspid Stenosis.—The *murmur* is diastolic (presystolic) in rhythm, and crescendo and rough in character, but is rarely as loud as the corresponding murmur of mitral stenosis. It

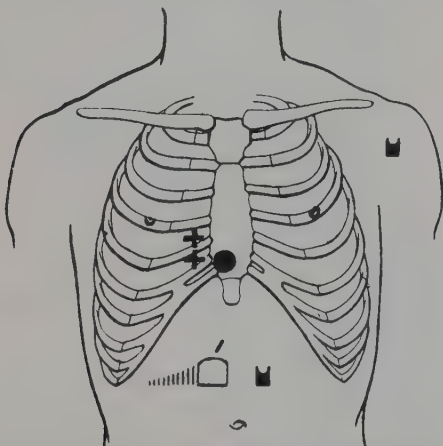


FIG. 233.—DIAGRAM SHOWING DISTRIBUTION OF PRESYSTOLIC MURMUR OF TRICUSPID STENOSIS.

is heard best at the xiphoid end of the sternum, and is of limited distribution (Fig. 233). A rough presystolic *thrill* has been observed in the same situation.

Tricuspid stenosis is usually associated with other valvular defects, and often with mitral stenosis. In some of the cases two maxima of the murmur have been observed, in the apical region, and at the xiphoid end of the sternum, but the two murmurs are often indistinguishable. It must be remembered that, in cases where the left heart is hypertrophied, the presystolic murmur of mitral stenosis may be well heard even over the sternum, and its thrill may be felt in the epigastrium.

Tricuspid stenosis is compensated by hypertrophy of the right auricle, but this is rarely sufficient.

The distribution of murmurs produced at the valves is a natural sequel to their anatomical position within the chest. *Any abnormality in distribution indicates that some abnormal*

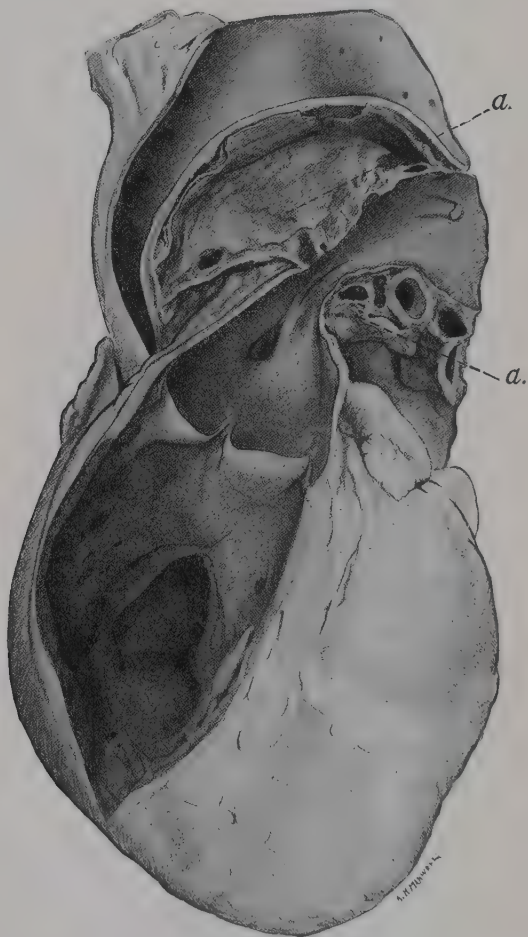


FIG. 234.—STENOSIS OF LEFT PULMONARY ARTERY FROM PRESSURE OF A MALIGNANT GROWTH (*a*).

factor has come into action. In a case of multiple cutaneous cancer, for instance, a secondary growth occurred in the mediastinum, and nipped the left pulmonary artery just after the bifurcation (Fig. 234), and a loud systolic murmur made

its appearance. It was well heard at the second left space, but it was louder beneath the left clavicle, and loudest over the left scapula (Figs. 235, 236), clearly showing that the

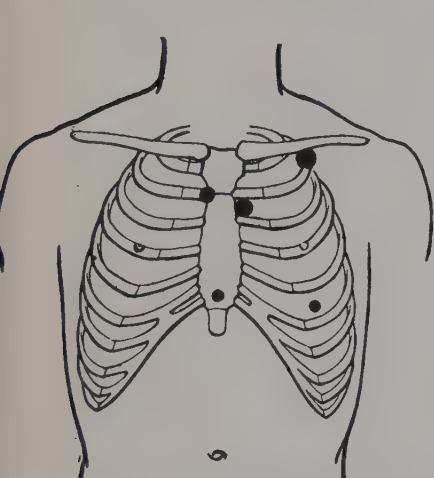


FIG. 235.—DIAGRAM SHOWING DISTRIBUTION OF SYSTOLIC MURMUR.

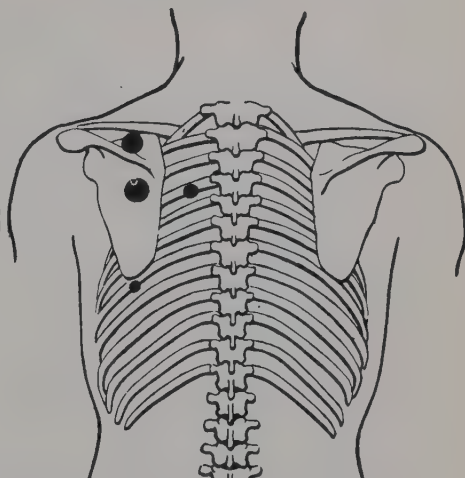


FIG. 236.—DIAGRAM SHOWING DISTRIBUTION OF SYSTOLIC MURMUR.

stenosis was situated some distance away from the pulmonary valve and the front of the chest. An aneurysmal dilatation, an intrathoracic tumour, or a pulmonary consolidation, may also lead to the conduction of murmurs in abnormal directions.

CHAPTER XIX

THE PROGNOSIS OF CHRONIC VALVULAR DISEASE

THE difficulty of forming an accurate prognosis in disease of the heart has been emphasized by many writers ; but though no one denies the frequency of error, a fairly accurate forecast can generally be made if all the facts of the case are carefully scrutinized. A wide outlook, however, is necessary, as the cardiac symptoms and signs are by no means the most important elements. An accurate prognosis, too, requires repeated and continuous observation, and can rarely be made on an isolated examination.

In the presence of *active* symptoms their progress from day to day forms, of necessity, the main basis on which an opinion can be formed. The size, the strength and rate of the pulse, the degree of œdema and hepatic enlargement, the frequency of the respirations and the grade of the dyspnœa or orthopnœa, the pallor or the cyanosis, the amount of urine excreted, the discomfort, the restlessness and the amount of sleep obtained, have all to be accurately recorded and compared from day to day. And it must ever be remembered that the “accidents” of valvular disease, produced by embolism, occur most frequently in the presence of general symptoms of cardiac distress, and may, in a moment, alter the whole aspect of affairs. But embolism is by no means invariably fatal, and we have seen patients live in comfort for several years after the occurrence of a cerebral embolism or a pulmonary infarct, though this is certainly unusual. A woman, aged forty, was recently in our wards suffering from cardiac failure consequent on mitral disease and auricular fibrillation. Four and a half years previously her right hand had suddenly become painful and cold and powerless, and gangrene followed, which necessitated amputation through the forearm. The radial and brachial pulses could not be felt before operation, though

the subclavian artery was pulsating, and the condition was apparently the result of an embolic block associated with organic mitral disease of rheumatic origin. She had been able to perform her household duties until a few months before her second admission into hospital.

Of isolated symptoms, two are particularly important and of evil omen. Few patients recover who display distinct jaundice with an enlarged, congested liver, or who lie in the trough of the bed, with lack-lustre eyes, completely indifferent to their surroundings.

Sir William Broadbent¹ laid down a useful scheme of inquiry on which the prognosis should be based in those cases in whom the urgency of the symptoms is not immediate. To this we have made some additions. The nature, the site, and the grade of the lesion; the rhythm of the heart; the presence or absence of associated disease in other viscera; the degree to which compensation has been effected; the cause of the failure which has brought the patient under observation, have all to be taken into account, as well as the age and sex of the patient and his social circumstances and habits.

1. *The Nature of the Lesion.*—The most favourable cases are those in which the mitral valve has become incompetent as the result of anæmia or debility, if the cause can be removed and its recurrence prevented. The corresponding incompetence of the tricuspid valve is more serious, for it rarely produces symptoms in the absence of organic disease of the lungs or left heart, and the cause of its occurrence consequently persists. A congenital defect is not progressive, but the majority of patients die in early life in consequence of the disturbance of the circulation, or, if they survive, are essentially weaklings. Valves which are so deformed are peculiarly prone to be affected by acute inflammatory processes. In patients, however, who have reached maturity with good physique and nutrition, and absence of any evidence of cardiac distress, the outlook is favourable, but though many cases have been recorded who lived to thirty and forty years of age, the records are less numerous in the fifties, and we can find but few over sixty. Of our cases, only two as yet have reached forty.

A chronic valvular lesion due to a long antecedent rheumatic

¹ *Heart Disease*, Lond., 1897, 67-87.

infection is unlikely to be progressive unless the exciting cause recurs, and has, in consequence, a good prognosis; but the age of the patient must be taken into account, for rheumatism often recurs in the 'teens and twenties, though less frequently after thirty. A syphilitic lesion may become stationary if it is recognized and treated. The progressive lesions are unfavourable. Acute endocarditis can only be gauged by the progress of the case and the degree of the compensatory changes in the size of the chambers. The occurrence of symptoms of cardiac failure is always a serious sign, particularly if they obtain during the febrile stage. The degenerative chronic lesions are necessarily progressive, though the rate of progress varies in different cases. Patients rarely live for five years after symptoms have once made their appearance.

2. *The Site of the Lesion.*—The possibility of sudden death in patients who are the subject of valvular disease has rarely to be considered, so long as the patient is not confined to bed, though it is by no means uncommon in cases where serious symptoms are already present. Sudden death, according to Kisch, is uncommon before the age of thirty, and is most frequent between fifty and sixty. It is apt to occur in fat people with general arterio-sclerosis, and in patients with fatty dilated hearts and mitral or aortic incompetence. It is not uncommon in aortic aneurysm, in cases with wholly irregular, or infrequent, pulses, in fat persons who suffer from angina, and in emphysematous patients who have manifested symptoms of cardiac weakness.¹

The causes of sudden death are thus related to myocardial rather than to valvular lesions, and are often the result of coronary disease. We have notes of 7 cases in which death ensued without warning, and in 5 the heart was ruptured. In the other two the coronary arteries were blocked by emboli, which were derived in one case from the apex of a dilated left ventricle, and in the other from fungating vegetations upon the valves.

Of the valvular lesions, aortic regurgitation is that which most often ends suddenly. The strain upon the left ventricle

¹ It has been suggested that the occurrence of *ventricular* fibrillation may be the cause of sudden death in cases where the *auricles* are fibrillating. One of us has witnessed such an occurrence (*see p. 194*). In experiment such a sequel is not uncommon.

is great if the regurgitation is extreme, and it occurs during diastole, when the ventricle is "defenceless" as it is not in contraction, and a fatal syncope may, in consequence, occur. In aortic disease, too, the coronary arteries are more frequently damaged than in mitral disease, and the muscle is thus more likely to be abnormal. In mitral stenosis embolism is relatively frequent, and may occasion sudden death. The experience at St. Mary's Hospital may be quoted: 4 out of 38 cases of aortic regurgitation died suddenly, but only 1 of 53 cases of mitral stenosis, and 2 of 44 cases of mitral incompetence, and in the latter serious symptoms were already present. In aortic stenosis (11 cases) there were no sudden deaths.

The most serious valvular lesion is disease of the tricuspid valve. It is uncommon unless in the form of "functional" insufficiency, secondary to left-sided heart disease, or affections of the lung, but as compensation has to be effected by the right auricle, its degree is usually insufficient, and it is rarely maintained for any length of time. Tricuspid regurgitation, aortic regurgitation, mitral stenosis, aortic stenosis, mitral regurgitation, in order, probably represents fairly accurately the relative danger of the lesions.

It is impossible to give figures, for the lesions are rarely comparable. For example, the age at death in mitral disease following acute rheumatism in early life cannot be contrasted with the age at death in mitral disease the result of chronic endocarditis which ensued after fifty. Balfour's case, where the patient had mitral incompetence for sixty-six years, had the longest duration which we can find recorded.

3. The estimation of *the degree of the lesion* has already been considered.

4. *The Rhythm of the Heart*.—We had hoped to have been able to give definite figures as to the frequency with which irregularities occur in mitral and aortic disease in the presence of symptoms, but our records are still scanty. In the mitral series (102 cases), auricular fibrillation occurred in 19 cases, paroxysmal tachycardia in 5, extrasystoles in 2, and heart-block, nodal rhythm, and an undecipherable irregularity in 1. In the aortic series (58 cases), extrasystoles occurred in 9, auricular fibrillation in 2, paroxysmal tachycardia in 1, and nodal rhythm in 3. But the records are imperfect, and

irregularities were more frequent than the figures suggest, though the large majority of patients had a regular pulse. Death may occur, in mitral stenosis, without any alteration in the rhythm, even in cases where the valvular lesion is considerable and of old standing. A woman, who was under the charge of one of us when house physician in 1895 in Dr. Tennent's wards, was admitted into our wards in 1912 for a few weeks before her death, at the age of forty-four. The mitral lesion had existed for seventeen years at least, and probably for longer, and the right heart was permanently enlarged; but notwithstanding the repeated occurrence of pulmonary infarcts, the cardiac rhythm remained regular, and the auricular wave persisted in the cervical curve.

The prognosis in cases with a regular cardiac rhythm is apparently less serious than in those with an irregular rhythm. But the other factors must be comparable if the comparison is to be exact, and as a permanently irregular rhythm is generally due to myocardial changes, the difference may be merely due to the greater incidence of the latter. From the practical point of view, however, the distinction is important.¹

¹ TABLE SHOWING THE MORTALITY IN CASES WITH A REGULAR AND AN IRREGULAR RHYTHM OF THE HEART.

	All Cases.	Rhythm Regular.	Rhythm Irregular.
Mitral disease	102	73	29
Death in	32	15	17
Per cent.	31.4	20.5	58.6
Aortic disease	58	43	15
Death in	17	11	6
Per cent.	29.3	25.6	40

	Mitral Disease.	Aortic Disease.
Auricular fibrillation	19	2
Death in	11	—
Extrasystoles	2	9
Death in	2	2
Paroxysmal Nodal tachycardia ..	5	1
Death in	2	1
Heart-block	1	—
Death in	—	—
Nodal rhythm	1	3
Death in	—	—

D. MACDONALD.

5. *The Presence of Disease in other Viscera.*—We have already indicated the frequency with which cardiac valvular disease is associated with lesions in other viscera, the lungs, the arteries, and the kidneys being most often involved. The presence of other lesions always intensifies the gravity of the prognosis. In young people they occur as a result of the cardiac lesion or irrespective of it, but in elderly persons they are frequently the cause of the valvular disease, and the recognition of the cardiac element alone leads to an erroneous idea of the case; for in many patients the cardiac flaw is not very material, and the progress is dependent upon, and the symptoms are due to, the other lesions. In comparable cases, of course, the prognosis is better in the purely cardiac group. Patients with cardiac disease and hepatic cirrhosis rarely live long after the latter has been recognized. Notable emaciation is always of serious import.

6. *The Degree of Compensation.*—Sir James Mackenzie has emphasized the well-known, but oft-forgotten, aphorism that the integrity of the cardiac muscle is the most important factor in valvular disease, and that, so long as the cardiac work is carried on easily, the exact degree or nature of the valvular flaw is immaterial; in short, that “the proof of the pudding is in the eating.” With this every one will agree, and so long as the ordinary business of life is conducted without discomfort, the immediate outlook is satisfactory.

It is difficult to estimate the reserve power of the heart, and it can only be gauged by repeated experiment as to the amount of physical exertion which can be undergone without discomfort. Even here a fallacy is at once apparent, for all of us who have entered or passed the forties, the forties which are so often as stormy in life as they are at sea, are well aware of the necessity of taking things quietly at the beginning of our annual holiday, and an amount of exertion which can be indulged in with impunity, or even with benefit, towards its conclusion, may be badly borne at its beginning.

In a general way a conclusion may be reached. If a day's golf or shooting is not accompanied by undue weariness or breathlessness, if business activities entail no special disability, and if a hill can be surmounted with ease, the cardiac reserve is ample, and little apprehension need be felt. Conversely,

if a slow walk on the flat is the only comfortable mode of progression, if œdema appears in the ankles at night, and if breathlessness ensues on walking upstairs, the reserve is slight and immediate trouble may be anticipated. In the consulting-room, similar experiments may be tried, and the results of hopping round the room, or walking upstairs, may be investigated. The behaviour of the pulse on exertion is often a useful guide. Its rate, when the patient is recumbent, is normally some seven to fifteen beats per minute less than when he is standing up. In the milder forms of insufficiency the difference is greater, or no fall in rate ensues when he lies down. On exertion, in healthy individuals, the pulse rate at first increases, but rapidly regains its former rate when the patient is again seated. The majority of normal persons regain their usual pulse rate within a couple of minutes after walking upstairs, but in myocardial insufficiency the period may be delayed for four or ten, or even thirty, minutes. In exceptional cases the pulse rate may be slowed by exercise. This is generally due to some of the beats becoming imperceptible to the finger, though it is said to result in exceptional instances from true slowing of the ventricular contractions. The occurrence is, in valvular disease, a sign of evil significance. But, in cases of so-called D.A.H., slowing of the pulse rate, after exertion, is as a rule the signal of commencing improvement.

7. *The Cause of Failure.*—In patients who come under observation on account of symptoms of cardiac weakness, the precise cause of the failure is an important element in the prognosis. The symptoms may be due to the activity of some new factor outwith the heart itself, to a progressive lesion of the valve, or to the development of some pathological condition of the cardiac muscle.

Extrinsic factors are numerous. Physical exertion, whether short and severe, or moderate and prolonged; bronchitis, pneumonia, etc., throwing extra strain upon the right heart; a gastro-intestinal upset or an alcoholic debauch; an attack of influenza; anæmia or debility; pregnancy and parturition; a mental shock or business worries, so often precede the onset of cardiac symptoms that their relationship to it can hardly be in doubt. Progressive lesions occur both in acute

endocarditis and in the degenerative chronic forms, and though usually gradual in their onset, may be sudden, if, for example, a cusp ruptures suddenly. Fatty overgrowth and degeneration and fibrosis of the myocardium are usually of insidious origin, but a sudden blocking of a coronary branch may produce symptoms with great rapidity.

The prognosis in the individual case varies accordingly. The possibility of a future pregnancy can be prevented; the patient may become teetotal; the occupation may be changed, or excessive work and strain may be avoided; and climatic change may retard the recurrence of pulmonary disease. The fatty overgrowth may be reduced by the cessation of "indulgence, indolence, and intemperance"; an anæmia may be overcome; while but little can be effected in checking the rapid progress of an acute endocarditis, or the insidious progression of the more chronic lesions which are affecting the valves or the coronary arteries.

In other cases failure is due to the inception of a new cardiac rhythm, auricular fibrillation, heart-block, paroxysmal tachycardia, etc. The prognosis in this event is always serious unless the progress of the case reveals the individual tendency towards convalescence. It should, however, be borne in mind that none of the abnormal rhythms are necessarily incompatible with life, which may be prolonged over a period of years.¹

8. *The Age of the Patient.*—The age of the patient must be taken into account. There is a maxim in our assurance office that in youth the family history, in adult life the habits, and in later years the circumstances, of proposers should be

¹ J. K. Rennie and one of us have examined our series of *post-mortem* records of cases of chronic valvular disease from this standpoint. The cases numbered 81, the mitral valve alone being affected in 37 instances, the aortic alone in 17, both in 21, and several valves (including the tricuspid) in 6. *Thirty-seven of these patients died from the results of an acute re-infection of the valves.* Of the remaining 44, 21 died from cardiac failure of unknown origin, 12 from pulmonary complications, 4 from cerebral causes, and 1 from starvation, otitis media, exertion, acute rheumatism, purpura, paroxysmal tachycardia, and heart-block.

The cause of failure is thus most commonly (45 per cent.) a fresh acute, or subacute, infection of the valves.

closely scrutinized ; and the maxim, on the whole, holds good with regard to the prognosis in cardiac disease. A family tendency to rheumatism, habits of excess, whether at work or at play, in food or in drink ; the anxieties of straitened circumstances, are all necessarily untoward elements in the case.

Muscular hypertrophy in early life is easily produced, and is generally ample ; but the probability of recurrence of a rheumatic infection is considerable in the 'teens and in the twenties, when it is most often accompanied by a fresh endocarditis. If growth is incomplete, its extra strain is badly borne, and a considerable lesion is generally followed by a stunted development and a poor physique. In elderly persons muscular hypertrophy is often limited in the case of the heart, as it is in the systemic muscles, and compensation may be effected with difficulty. Other viscera, too, are apt to be damaged, and the lungs, the kidneys, or the arteries are frequently abnormal. The nutritional demand, on the other hand, is lessened.

The condition of the ribs is probably important. In childhood, when they are soft and yielding, the contour of an enlarged heart may be mapped out on the front of the chest ; but in elderly people, protrusion is unusual, and the heart can only enlarge at the expense of the other organs within the thorax. On the whole, early adult life is the least unsatisfactory age, for the strain of growth is past, and the probability of a fresh infection is less, while muscular hypertrophy is likely to be ample, and the other viscera are probably sound. The unsatisfactory feature is the activity of manhood, and a physiological mean must be carefully enjoined.

9. *The Sex of the Patient.*—The influence of sex is not very considerable ; for, while women as a rule escape extremes of heat and cold, and the strenuous physical exertions to which men are so often exposed by reason of their special occupation, the gain is counterbalanced by their maternal functions. Pregnancy, in our experience, is always a dangerous experiment in women who are the subjects of chronic valvular disease. Failure often occurs in the later months or following child-birth, during lactation, though, curiously enough, it does not seem to be particularly related to the accouchement.

It may be due to general debility or to a fresh endocarditis, or to a renal complication. The latter two are the most frequent causes. The cases already recorded instance the connection (*see* p. 225).

10. *The Social Circumstances and Habits of the Patient.*—The habits and social circumstances of the patient are important. Arduous employments are, of course, always injurious to patients whose cardiac reserve is limited by valvular disease ; but they may be the only means of obtaining a livelihood ; and those in receipt of a weekly wage are rarely able to secure the just medium of mental and physical work that is so desirable. The prognosis in hospital cases is, in consequence, less favourable than in private patients.

CHAPTER XX

MYOCARDIAL FAILURE WITHOUT VALVULAR DISEASE

IN chronic valvular disease the cardiac work may be carried on for many years without any symptoms of cardiac distress, which only occur when the muscle becomes unable to accomplish its necessary task. The failure may be due to many causes : the action of a *new* factor, such as bronchitis, throwing an extra strain upon the heart ; an increase in the degree of the valvular lesion to such a point that the reserve becomes minimal ; or the occurrence of pathological changes in the muscle itself.

Myocardial disease may, however, occur in the absence of valvular disease, and occasion very evident symptoms of cardiac distress. But, though the clinical picture may be purely cardiac, pathological examination shows that the lesions are rarely, if ever, confined to the heart. The granular degeneration of the acute infections is accompanied by cloudy swelling in other viscera. Fatty degeneration is associated with fatty degeneration elsewhere. Fatty infiltration affects the whole body, not the heart alone. The fibrosis of arterial lesions is rarely limited to the area of the coronary arteries.

The symptoms of myocardial weakness are similar to those which occur in valvular disease, when compensation has broken down, namely, *shortness of breath, palpitation, thoracic discomfort or pain, and œdema.*

In the *infections* the symptoms of cardiac failure are masked to some extent by those of the toxæmia and of any local lesion which may co-exist. But the increasing weakness, breathlessness, and cyanosis, which obtain in serious cases, own, in part at any rate, a cardiac cause. Coldness, pallor or congestion of the extremities are not infrequent, and faintness

or intense prostration may occur on even trivial exertion, such as sitting up in bed. Œdema is somewhat rare, and most frequently the result of venous thrombosis, but it may be purely cardiac in origin, and though usually trivial, is occasionally widespread and extreme.

A married woman, aged thirty, who had nursed her fourth baby for nine months, gradually began to find that her housework was becoming exhausting, and that she became short of breath, and felt her heart beating on very slight exertion. Vomiting set in, and continued for several weeks. She lost flesh rapidly, and became pale and sallow in appearance. On admission to hospital, she was very weak and exhausted, and notably emaciated and pale, the red cells only numbering 1,060,000, with a hæmoglobin value of 20 per cent. Films showed a well-marked megalocytic reaction. A few days afterwards she became fevered, and a consolidation gradually involved the left lung. With the onset of the pneumonia exhaustion became extreme and incontinence ensued, and coincidently œdema, which on admission had been trivial in amount, became universal and considerable. The pulse was now very small and soft and frequent, and the cardiac sounds were short and weak. Three weeks later the consolidation was resolving, the œdema had disappeared, and the pulse, though still small and soft, approximated to the normal rate. She eventually made a good recovery, and four years later was strong and well.

During *convalescence* from acute infective diseases, on the other hand, the usual symptoms of cardiac weakness, shortness of breath on exertion, and œdema, are extremely common, and are sometimes very persistent, though in the vast majority of cases they disappear rapidly with improvement of the general health. Cardiac discomfort, palpitation, tachycardia, and pain may also occur, and may suggest from their continuance the possibility of serious mischief, though complete recovery is the rule, unless organic disease is already present. The palpitation and tachycardia are extremely trying to the patient, and may continue for months. In the case of a medical friend, after an influenzal attack, even such trivial exertion as walking slowly upstairs induced profuse perspiration, with throbbing in the chest and head, while the pulse rate ran up to the neighbourhood of 200 per minute. A sensation of faintness is common, but actual syncope is rare.

Fatty Changes.—The fatty changes which occur in the heart are as a rule associated with disease elsewhere, and

give but little clinical evidence of a specific kind. Fatty degeneration is most common in severe anæmia, in chronic alcoholism, in emphysema, and in chronic disease of the kidneys, and the cardiac symptoms are only a part of a general disturbance. Fatty degeneration co-exists with granular changes in the infections, but its particular degree cannot be appreciated during life. In disease of the coronary arteries it is merely a phase of an ischæmic atrophy. As an isolated lesion affecting the heart and the heart alone, it is of rare occurrence, although it is evident that cardiac weakness from fatty changes frequently occurs.

Fatty infiltration is as a rule only a part of a general tendency to obesity, the presence of which naturally suggests the nature of any existing myocardial weakness. But with obesity, again, renal and arterial disease are frequently combined, and the cardiac picture is, in consequence, obscured.

It seems probable that there is no real distinction between fatty infiltration and fatty degeneration, which differ in site rather than in nature. The quality of the fat in the two situations has been shown to be the same in animals in which the lesion has been produced experimentally, and fats which are abnormal to the species may be deposited, if a particular fatty dietary is adopted. Fatty infiltration, too, is invariably accompanied by a certain amount of fatty degeneration, though the converse proposition is by no means invariably true.

In the exceptional case fatty infiltration of the muscle occurs without the epicardial or the general fat being excessive. The causes of this are not yet recognized ; but the fact is of importance, as the nature of the cardiac weakness is apt to be misinterpreted. A striking example of this once came under our observation. An old man who was convalescing apparently satisfactorily from a mild attack of smallpox, somewhat suddenly became weak and stupid, and passed rapidly into coma, dying within forty-eight hours of the onset of serious symptoms. There had been no symptoms previously which suggested any special cardiac weakness, and his general nutrition seemed good, and there was no special obesity. The heart, however, showed extreme fatty infil-

tration, particularly upon the right side, where fatty deposits were visible even on the endocardial surface ; and the muscle cells were also grossly affected. His arteries generally were atheromatous and hard, and the minute arterioles on the right side of the heart were affected to a greater degree than on the left.

The symptoms of fatty change in the heart do not differ from those of cardiac weakness owing to other causes, and the evidences of cardiac insufficiency are similar. The heart, too, from associated lesions may be large or small ; the pulse may be hard and full, or small and soft ; and the murmurs indicative of valvular disease, or alterations of the sounds from high blood-pressure or a hardened aorta, may be absent or present in particular instances. The thickness of the parietal fat in general obesity may interfere with the examination of the heart, and the character of the pulse may be the sole trustworthy guide. Frequently one is scarcely able to hear the cardiac sounds, though the pulse is full and hard, with a pressure decidedly above the normal.

Disease of the Coronary Arteries.—The relative frequency with which individual arteries are diseased has been investigated by several writers. Every one is agreed that the aorta is most frequently affected, but while Lobstein states that the splenic and the femoral arteries are more often diseased than the coronary arteries, and Rokitsky gives precedence as well to the iliac vessels, Huchard¹ considers that the arteries of the heart are those which are most frequently abnormal. For this predominance several reasons, he states, are apparent. Their high origin in the aorta where the blood-pressure is at a maximum, their mainly muscular texture, their numerous and acute curves, their constant activity in supplying blood to tissues constantly in action, etc., all tend to render them unduly liable to disease ; while their relationship to the regularly contracting heart, however well protected they may be in their bed of fat in the sulci, exposes them constantly to jars and contusions. The exact reason for their involvement, however, is of less moment than its frequency, and the importance of their integrity cannot

¹ Huchard, H., *Malad. du cœur*, Paris, 1899, i., 205.

be over-estimated, for upon this the nutrition of the heart depends.

Myocardial disease is rarely, if ever, universal, and the local distribution is dependent upon local and most commonly vascular causes; and, as we have seen, atrophy, fatty, and hyaline degeneration, necrosis and fibrosis, may all be due to arterial disease. In arterio-sclerosis the arterial change is widespread, and the existence of coronary lesions can be appreciated by recognition of the existence of the change in the peripheral vessels; but in focal disease the distribution is often erratic and apparently whimsical, and the visceral vessels may be seriously affected without any evident change in the arteries which are accessible to examination. It is this factor which makes prognosis in cardiac disease so imperfect, for our ignorance of the condition of the coronary arteries renders it impossible to assess the most important factor at its true value, and the "happy guess" sometimes turns out to be wrong. An embolus occludes the lumen, a narrowed vessel becomes completely blocked, and the cardiac contractions suddenly and for ever cease.

Occlusion of the coronary arteries may be of gradual or of sudden onset, and may affect the superficial "rings" or the perforating branches. The anastomotic connections of the rings may prevent any serious damage to the muscle if the occlusion is sufficiently gradual, and several cases in which this occurred have been recorded. But rapid closure prevents adequate compensatory arrangements, and the muscle which is concerned in consequence dies. Even in gradual closure adequate anastomotic developments are exceptional, and the muscle suffers in greater or less degree. Closure of a nutrient artery almost always produces necrosis.

The changes which are found *post-mortem* thus differ in accordance with variations in the rapidity of the closure and in the site of the affected vessel. And while at one end of the chain, where the anastomotic developments are sufficient, the cardiac muscle may be perfectly normal, and at the other, where a large vessel has been suddenly closed, may show no microscopic lesions, because sudden death has prevented structural changes, the intervening links exhibit every intervening grade in extent and severity, and the clinical symptoms

follow suit. Death may ensue in a moment, or after an illness of many months, and serious failure may pass away if satisfactory anastomotic developments eventually follow.

Experimental ligation of a coronary artery or of one of the main branches is often rapidly followed by death, but this is by no means invariable, and the animals may live for hours, days, or even weeks without the appearance of any notable symptoms beyond a transient irregularity of the cardiac action; and a case has been recorded (Pagenstecher, quoted by Hirschfelder) in which the right coronary artery of a man was ligatured without harm.¹ Clinical and pathological observations show similar results, and the changes which are found even in cases of sudden death often show that coronary occlusion must have occurred some time previously.

If the occlusion is sudden and complete, immediate death may ensue. A woman, aged thirty-two, underwent a somewhat tedious operation for gall-stones, and on the next morning was very cheery and well, but about noon died suddenly, without any warning. On examination, a large mobile embolus was found to block completely the descending branch of the left coronary artery, which was otherwise normal. The embolus had arisen from a clot at the apex of the left ventricle, which was evidently of some age, and must have existed prior to the operation. But in other cases—*e.g.*, in rupture of the heart—necrosis has occurred previously, and the fatal issue, though abrupt, takes place subsequent to the occlusion. An insane woman, aged seventy-eight, who went to bed in her usual health, was found dead in the morning, rupture of the left ventricle having occurred on its posterior and upper aspect. The tissues at the site of rupture were soft and friable and necrotic, and the transverse branch of the right coronary artery was found to be thrombosed for nearly an inch. The inflammatory reaction in the periphery of the infarct had so weakened the tissues that the endocardial blood had gained entrance to the muscle, and, forcing its way along the tract of the infarct with successive contractions of the heart, ultimately reached and perforated the pericardial covering. Infarcts, however, are not necessarily fatal, and may produce little sign of cardiac failure. A woman, aged fifty-seven,

¹ Hirschfelder, A. D., *Diseases of the Heart*, 1918, 379.

died after an illness of five days' duration, during which the symptoms were wholly cerebral, the result of streptococcic abscesses in the cerebellum, and meningitis. There were no signs of special cardiac embarrassment, even during the last twenty-four hours of life, though the mitral valve was extensively disorganized by recent ulceration superimposed upon a valvular defect of old standing, and a large infarct was present at the apex of the left ventricle.

Myocardial Fibrosis.—If the coronary obstruction is gradual, the onset of symptoms is necessarily insidious and the course of the disease is essentially chronic. This is the rule in aneurysm of the heart; for sudden occlusion, if of any extent, causes sudden death or infarct, and in the latter case is more usually associated with rupture than with fibrosis.

A policeman, aged forty-nine, caught cold one January, which did not, however, necessitate his ceasing work; but when the cough had gone, he noticed that he was somewhat breathless on exertion, and experienced a sensation of tightness in the epigastric region. In April the cough recurred, and was now accompanied by a scanty expectoration, and he felt so unwell that he went on holiday to the Highlands. Here he improved to some extent, but was still troubled with breathlessness, especially when walking uphill. On his return home he still felt unwell, but he continued to work until September, when his feet for the first time became swollen. At night, too, he was very short of breath, and had to sit up in bed, and slept badly. His weight had decreased, and he was two stones lighter than usual. On admission to hospital, the oedema, which was general, was not great, but the right chest was half full of fluid, and he was very breathless and orthopnoic. After the fluid had been removed, his symptoms rapidly abated. The heart was large, especially on the right side, and the sounds were short and distant, but pure. The pulse was regular, but small and weak, and numbered about 100, and even when his other symptoms had disappeared still ran between 90 and 100. After a month's residence in hospital he was sent to a convalescent home, but the exertion of the journey caused the symptoms to recur, and he had to return to the ward in three days. His progress afterwards was continually downhill. The oedema and effusions steadily increased, and he died with slow cardiac failure a year after the commencement of his illness. *Post-mortem* examination showed that the failure was due to an extensive fibrosis of the apical third of the left ventricle and of the interventricular septum, which had followed occlusion of the descending branch of the left coronary artery.

A married woman, aged sixty-five, was admitted into hospital

suffering from breathlessness and general œdema of about three weeks' duration.

She had always been healthy, and had never had any serious illness previously, but for the two preceding winters had been liable to bronchitis, and had complained of palpitation. Breathlessness on exertion and swelling of the feet at night had made their appearance about a year before admission, but the onset was insidious, and the symptoms did not interfere with her household work until three weeks before admission, when she was forced to take to bed. She became excited and orthopnœic, and the œdema of the legs became so great that the skin gave way and serous fluid exuded.

On admission she was mildly delirious, and unable to lie down in bed, the dyspnoea being extreme. Œdema was universal and great, and the sores on the legs were foul and evil-smelling, and exuded a thin serous discharge. The pulse was small and very irregular, numbering about 100. The heart was evidently enlarged. The second pulmonic sound was emphatic, while the first sound at the apex was short and sharp, and occasionally followed by a soft blowing murmur. The urine was scanty, and contained albumin in small amount. Several syncopal attacks occurred, and she died suddenly on the third day after admission.

On *post-mortem* examination, the pericardial sac was found to be almost wholly obliterated by firm adhesions. The heart weighed 23 ounces. A large aneurysmal dilatation was present on the posterior wall of the left ventricle at the junction of the upper and middle thirds, its wall being apparently wholly composed of fibrous tissue and notably thinned, measuring only about one-third of the thickness of the ventricular wall in the vicinity. The main trunk of the right coronary artery was impervious from a short distance after its origin. The aortic and mitral valves were thickened and atheromatous, and a recent acute infection of the mitral valve (perhaps the result of the sepsis of the legs) was also present. The kidneys were cirrhotic. The origin of the pericarditis could not be explained by any antecedent illness, and it seemed to be probably secondary to the myocardial lesion.

The evidences of cardiac weakness, even in aneurysm of the heart, may be extremely slight, and cases have been recorded where they were wholly absent even for long periods.¹

A clerk, aged sixty-eight, was admitted into hospital suffering from chronic uræmia of some months' duration. He was mildly but constantly delirious, and could give no information as to his past history. He was very restless and irritable, and the breathing was Cheyne-Stokes in character, apnoea lasting for about half a minute, and being associated with slight twitching of the fingers, contracted pupils, and

¹ Wilson, C., *Lancet*, 1919, ii., 199.

an increased pulse rate. Death ensued in coma. On *post-mortem* examination the kidneys were found to be cirrhotic, and the heart was greatly enlarged, weighing $21\frac{3}{4}$ ounces. The left coronary artery was considerably narrowed, and the anterior wall of the left ventricle was fibrous and thinned and distended, the cavity being filled with *ante-mortem* clot. Three weeks before his death, however, when the lesion must have been considerable, the brachial blood-pressure measured 160 mm. systolic, 90 mm. diastolic, while the pulse rate tended to be infrequent (60) until the near approach of death, when it reached 100, and became irregular. There was minimal œdema in the legs on admission, but it soon disappeared and did not recur, so that practically the whole evidence of cardiac insufficiency lay in the symptoms of renal inadequacy.

It seems certain from the pathological evidence that gross infarcts of the myocardium may occur, and yet ultimately healing may take place. Such an occurrence would explain some of those temporary cardiac failures which are occasionally met with in elderly people, in whom serious failure ensues more or less abruptly without obvious cause, and ultimately passes off. In the following case such a state of matters seemed extremely probable, but the crucial confirmation is lacking.

A carter, aged thirty-two, who presented many of the signs of incipient locomotor ataxy, noticed that his feet had become swollen without any obvious cause. The œdema rapidly increased, and a week later a slight cough, accompanied by a muco-purulent sputum, supervened, and the abdomen became swollen and tender. When admitted into hospital, a fortnight after the onset of the illness, the œdema was universal and considerable, the penis and scrotum being so much swollen that micturition was difficult. The liver was enlarged and tender, and the heart was large, but the sounds were pure, the first sound at the apex being short and sharp, while the second aortic sound was emphatic. The arteries were thickened and tortuous. Three weeks later the œdema had disappeared, and the patient's discomforts had ceased, and his convalescence was uninterrupted. The failure seemed to be purely myocardial, and the absence of all the usual causes of failure and the presence of widespread arterial disease suggested that it resulted from disease of the coronary arteries. The area involved, however, was sufficiently small for complete recovery to be possible.

Acute Myocarditis is a not uncommon cause of cardiac failure. As already mentioned (p. 222), it is a practically constant accompaniment of acute endocarditis, and often of pericarditis. It may occur, too, in acute infections which

are not accompanied by endocarditis. It does not, however, occur as an isolated lesion, but only as a part of some infective disease. In such cases its presence may be suspected if the cardiac failure seems in excess of that usually associated with general symptoms of like intensity.

The Physical Signs of Myocardial Failure

The *physical signs* in myocardial disease are often altered by coincident disease. The apex impulse may be displaced by renal disease, or obscured by emphysema. The sounds may be obscured by murmurs of valvular origin. The relative intensity of the second sound may be altered by arterial or pulmonary lesions. Displacement of the heart may occasion pulsation in abnormal sites, or conduction of the sounds in abnormal directions.

In these cases it is often helpful to *contrast the character of the apex impulse, the quality of the pulse, and the character of the cardiac sounds*. If one of these is not proportionate to the others the prognosis should be based upon the more serious sign. In renal disease, for example, the apex impulse may be more forcible than normal and the pulse of high pressure, while the character of the cardiac sounds suggests weakness. In these cases the first sound is rarely sharp or loud, and generally dull, distant and faint. Its termination may be indistinct, though no definite murmur is present. The contrast between these three factors is sometimes extremely striking. An old man, dying slowly of cardiac failure, still presented a couple of days before his death, at a time when œdema was widespread and extreme, a slow, deliberate, powerful apex impulse, though the first sound was almost inaudible.

The relative intensity of the sounds over the right and the left ventricle should also be compared, for cardiac strain may be evident in one place and not in the other. In pneumonia the difference between the two is often notable.

The physical signs of increasing cardiac weakness are well seen in *acute infective diseases*, in particular those of general type, such as typhus fever, for notable changes may occur within a few hours, and so be readily appreciated.

The *apex impulse*, as the weakness increases, loses its

punctate character and becomes more diffuse, while its force and duration diminish until it wholly disappears. It can, however, often be recovered if the patient lies upon his left side, but eventually, if the weakness is extreme, it fails altogether. The *area of dullness* is rarely notably altered, but slight variations in size can sometimes be determined. The *auscultatory signs* are more distinct. The first sound at the apex loses its special emphasis and becomes less loud and more distant, while its quality alters and it becomes shorter and sharper, approximating in character to the second sound. If the heart is beating frequently, the sounds closely resemble the tic-tac of a watch or the foetal heart. Less commonly the character of the sound is not altered, but it becomes weaker and shorter. The change is best appreciated at the apex, where the first sound is normally loudest and the most emphatic; with its weakening the second sound becomes relatively loud and so apparently accentuated. The sounds at the base are similarly affected, but as the second sound has the emphasis here, the alteration is less readily recognized. The first sound may finally disappear at the base, though still audible at the apex. Eventually it may be lost everywhere. The character of the sounds at the apex and the sternum may be found to differ, particularly if a pulmonary lesion (*e.g.*, pneumonia) throws a special strain upon the right ventricle.

The *pulse rate* as a rule becomes more frequent with increasing weakness. The amplitude of the wave is not at first affected, but its force and duration are lessened, and it becomes softer and quicker, and perhaps dicrotic. Eventually the volume lessens. The *rhythm* of the pulse is occasionally changed. An infrequent pulse in an acute illness is generally due to a coupled rhythm produced by recurring extrasystoles, the extrasystole being imperceptible to the finger, though it can be recognized on auscultation. Heart-block, auricular flutter, and auricular fibrillation may also occur, and we have observed them in pneumonia and acute rheumatism, though they are uncommon even in cases where acute endocarditis has ensued. G. Fleming has been kind enough to give us some statistics on this point from his experience in Ruchill Fever Hospital. Of 29 cases (diphtheria, 10; scarlatina, 9;

enteric fever, 7; typhus fever, 3), where routine tracings were taken from the second or third day of the illness, 2 alone showed extrasystoles, and these only occurred in 5 of 61 patients suffering from various fevers, in whom tracings were taken on account of some cardiac abnormality. Heart-block was detected in 1 case of diphtheria, in which the auriculo-ventricular bundle was subsequently shown to be involved in an inflammatory lesion; and a transient partial heart-block in one case each of diphtheria, enteric fever, and scarlatina.

CHAPTER XXI

THE TREATMENT OF CHRONIC CARDIAC FAILURE

THE general indications for treatment in cases where compensation has broken down are comparatively clear. *The heart must be relieved of all unnecessary work, and its nutrition must be improved. Symptoms which by their presence throw a strain upon the heart must be relieved. Cardiac stimulation may be required.*

1. Considerable relief is almost always experienced after the cardiac work has been reduced to a minimum by confining the patient wholly to bed, under the charge of an efficient nurse (Fig. 237). In no other way can the unnecessary work be so thoroughly removed, for the rate of the pulse is always increased when the patient is even sitting up, and if 5 beats per minute are saved, it means 7,200 beats in the twenty-four hours. The patient's comfort, however, must be consulted, for discomfort means restlessness, and restlessness entails work, and support by means of pillows or a bedchair is required in cases where orthopnoea exists. In the lesser degrees of failure, of course, such restrictions are unnecessary, and the patient may be able to sit quietly at home or to take a limited amount of exercise, but the desired result is attained most quickly by absolute confinement to bed. It is always easy to diminish restrictions, but it is often difficult to increase them. The rationale is simple: the expenditure must be curtailed until the overdraft is paid off, and when this is accomplished, must be rearranged on such a scale that the income is not exceeded.

The rest must be mental as well as physical, for mental worry and strain entail extra work to the heart, as the blood-

pressure is thereby raised and the pulse rate is often increased.

The return to ordinary habits, when convalescence is established, must always be gradual and should be closely supervised, so that any injurious results may at once be recognized. It is often helpful to have patients massaged mildly when they are still confined to bed and the effects can readily be estimated; and the general improvement in nutrition which is thus produced as a rule shortens the subsequent convalescence.

It is impossible to lay down absolute rules as to the period for which patients should be confined to bed, but it is never wise to allow them out of bed until all the symptoms are in abeyance, and the pulse rate is again normal, and the confinement should even then be continued for one or two or even three weeks longer. Some elderly people, however, bear severe restrictions badly, confinement to bed producing loss of appetite, sleeplessness, mental depression, etc., and it is sometimes wise in these cases to allow the amount of exertion to be determined by the patient himself rather than by his medical attendant.

2. *A priori* a weak heart requires more nourishment than a healthy one, so that in cardiac failure every effort should be made to insure an adequate supply; but the digestive disturbances which so often co-exist may render the task difficult. In serious failure the symptoms are frequently intensified by abdominal distension, vomiting, and flatulence; diarrhoea is uncommon. The dietary, in consequence, must be of a kind that throws little work upon the digestive organs. Carbohydrates and vegetables are generally contra-indicated on this account, for both are liable to produce flatulence, and

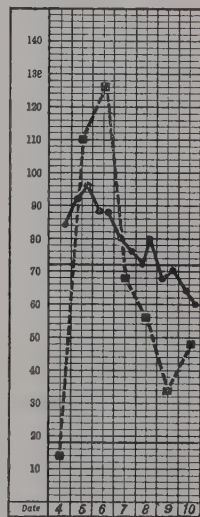


FIG. 237.—ARTERIO-SCLEROSIS; AURICULAR FIBRILLATION; CARDIAC FAILURE.

Dec. 4: (Edema universal and considerable.

Dec. 10: (Edema minimal.

- - - urinary output in ounces; — pulse rate.

Treatment: Rest in bed and a simple alkaline mixture.

while the latter notoriously entail extra work, the absence of oral digestion in serious cases is likely to disturb the proper digestion of the former. Milk is usually well borne in these cases, and should be given in small quantities and frequently (every two to three or four hours during the day, and every four or five hours at night). It may be predigested or diluted, or flavoured with weak tea, coffee, rum, etc. ; 3 or 4 pints may be given in the twenty-four hours. Benger's or Mellin's food can sometimes be added with benefit.

If the tongue is clean and the stools are normal, solid food may be given, small, lean, fine-fibred fishes, such as whiting, haddock, and sole, the breast of young birds, and eggs lightly boiled or poached, being the most suitable. Carbohydrates should be given in minimal amount and in dry form, toast, rusks, biscuits, etc., so as to ensure thorough mastication. The amount of solid food at first should be small, so as to avoid undue distension of the stomach. As the quantity of solid food is increased, the intake of milk may be correspondingly diminished.

The cooking of the food must be of the simplest kind, but as much variety as possible should be secured, so as to increase the psychic appetite ; and it may be remembered that a fried fish, *free of its breadcrumbs and fat*, is just as digestible as, and infinitely more appetizing than, one which has been plainly boiled or steamed. Soups, both clear and thick, are generally inadvisable, but stewed fruit (apples, pears) may be useful as a medium for the consumption of cream.

The intake of fluids requires regulation. In many cases dropsy is present, and it seems desirable to limit the intake of water ; but in many patients renal complications co-exist, and the blood-pressure is high, and the intoxication is best remedied by the administration of fluid in sufficient amount. The estimation of the blood-pressure is all-important from the point of view of treatment ; for if it is high, free elimination must be secured before any good result will be obtained. In young people, however, the kidneys are generally normal, and the intake of fluid may be restricted with benefit. The amount taken at meals should always be small (less than 10 ounces), so as not to interfere with digestion.

A similar distinction must be made with regard to the

consumption of table salt. In acute nephritis and in many cases of chronic nephritis the excretion of sodium chloride is deficient, it accumulates in the tissues, and to keep the fluids of the body isotonic attracts fluid to the parts. The resultant dropsy can be, in part at any rate, relieved by the withdrawal of salt from the diet. In cardiac cases with high blood-pressure sodium chloride retention frequently occurs, and its intake should, in consequence, be restricted. Less than a gramme of salt daily is required by a healthy individual, who, however, habitually exceeds this amount, sometimes to a very notable degree. Salt is present in bread and in almost all cooked dishes, if one excludes sweets from the list. Its use in bread and in cooking should be interdicted, and the amount used at table carefully restricted to less than the normal requirement of a gramme.

In elderly persons a sudden and complete change of diet is often badly borne, being apt to produce anorexia or indigestion of varying type and degree. The patient's habits in this respect should be considered, and a detailed list of the articles of food which are habitually consumed will generally be found to contain some elements which are relatively innocuous. The use of tea and alcohol, for example, should often be continued, though the amount or the quality may require to be altered.

Balfour¹ recommended the following "dry" dietary in cases of senile heart when there is anasarca:—

Breakfast.—A single slice of dry toast with no butter, and 4 ounces of tea infused for not more than four minutes, with cream and sugar.

Dinner.—Not more than the lean of two chops or their equivalent in fish or chicken; no vegetables; as much dry toast as may be desired, and $\frac{1}{2}$ ounce of spirits in 3 ounces of water.

Supper.—As much dry toast as is desired, and $\frac{1}{2}$ ounce of spirits in 3 ounces of water.

He did not consider that it was advisable that a patient in this condition should drink much, even between meals, and allowed only 3 to 4 ounces of hot water, sipped slowly, about an hour before each meal. A dietary so restricted is, however, rarely well borne for any length of time in chronic heart disease,

¹ Balfour, George, *The Senile Heart*, Lond., 1896, 252.

the restriction of fluids in particular leading to much discomfort; but the consumption of 2 pints of fluid in the twenty-four hours is generally tolerated.

The following diets are in use in our wards, and are generally useful. In private practice they have usually to be modified, but the general idea can be maintained. The chief meal should be taken in the middle of the day. The patient should rest quietly for at least half an hour after the larger meals, and for the same period before them, and efficient mastication must be insisted upon. Every effort should be made to ensure a sufficient variety in the foodstuffs, so as to encourage appetite.

"CARDIAC" DIET.

5 a.m. : Milk, 8 ounces; bread-and-butter, 2 ounces.

8 a.m. : Tea, 8 ounces; bread-and-butter, 6 ounces; fish, 4 ounces, or a lightly boiled or poached egg.

11 a.m. : Milk, 8 ounces, or milk and Benger's food, or a switched egg.

1 p.m. : Chicken, boiled or roast mutton, 4 ounces; bread, 4 ounces; milk pudding, 6 ounces.

5 p.m. : Milk pudding or tea, 8 ounces; bread-and-butter, 6 ounces.

7 p.m. : Milk, 8 ounces; bread-and-butter, 4 ounces.

The bread is stale and toasted. The diet is free from salt, which is added at the table if ordered. The total fluid may be restricted to 30 ounces. Milk pudding includes cornflour, ground rice, semolina, tapioca, sago, arrowroot, custard. Milk may be made more digestible by being citrated, "soured," or peptonized, and more palatable by the addition of weak, freshly infused China tea or coffee as flavouring agents; or may be given in the form of curds or junket.

"LIGHT" DIET.

5 a.m. : Milk, 10 ounces.

8 a.m. : Milk, or milk and weak tea, 10 ounces; bread-and-butter, 2 to 8 ounces; white fish, 4 ounces, or an egg lightly boiled or poached.

11 a.m. : Milk or Benger's food, 10 ounces.

1 p.m. : Chicken or white soup, 10 ounces; chicken or white fish, 4 ounces; potatoes, 2 ounces; vegetables, 1 ounce (cauliflower, cabbage, sprouts); bread, 1 ounce; milk pudding, 10 ounces.

5 p.m. : Milk, or weak tea and milk, 10 ounces; bread-and-butter, 2 to 8 ounces; white fish, 4 ounces, or an egg.

8 p.m. : Milk, 8 ounces. Cream, 10 ounces *per diem*.

This diet is salted.

3. The Treatment of Symptoms.—The relief of particular symptoms is often a necessary antecedent to any real improvement, for dropsy, pain or sleeplessness may of themselves prevent that rest which is so essential. In the slighter degrees of dropsy it may be sufficient to keep the bowels loose, but in the graver forms the fluid should be removed without delay, accumulations in the serous sacs being removed by a trocar and canula, and subcutaneous œdema by multiple puncture, or by the use of Southey's tubes. The punctures should be numerous, but separated from each other by an inch; two or three rows may be made on each side of the legs below the knees. The punctures should not be deep, merely through the skin, the needle being "guarded" by the finger and thumb $\frac{3}{8}$ inch from the point. With a sharp, broad surgical needle the stabs may be made with great rapidity and occasion little pain. Asepsis must, of course, be maintained by appropriate dressings.

The dyspnœa, which so frequently aggravates a patient's sufferings, is often extremely difficult to treat. It may be due to bronchitis, to even small amounts of fluid in the pleural sacs, to œdema of the lungs, or to toxic causes; but, however caused, may interfere gravely with sleep and rest. Much can be done by suitable bed-rests, etc., to make the patient comfortable when sitting up in bed, but in some cases a chair affords more ease, though in the latter case any œdema in the legs is generally increased. Oxygen is sometimes helpful. If there is fluid in the chest, it should be removed, even though but half a pint can be taken away; while bronchitis may be helped by the usual means. Œdema of the lungs can only be remedied by improvement of the cardiac condition. But in the toxic cases much relief may be obtained by the use of mercury and morphia, the former being given as blue pill or calomel in sufficient amount to secure two or three loose stools in the day, and the latter hypodermically and combined with atropine. The dose of morphia at first should be small (gr. $\frac{1}{8}$ to $\frac{1}{6}$), but it may be increased if well borne. It rarely occasions trouble, though care must be exercised if there is much secretion in the bronchi, or if the urine is scanty and highly albuminous. Heroin is a useful variant. A similar line of treatment is often useful in sleepless patients. Insomnia

is frequently accompanied by dyspnœa and by great restlessness, and is but rarely benefitted by the usual hypnotics. A dose of bromide in hot whisky may be sufficient in the milder cases, and may be backed up by chloral. Paraldehyde also is useful at times. In chronic cases, however, morphia is invaluable, and improvement is often rapid after two or three good nights' sleep have been obtained. In high-pressure cases W. Russell finds vaso-dilator drugs useful. Appropriate treatment may be required for cough, but the cough of irritation must be distinguished from that associated with profuse secretion; and while morphia is indicated in the former case, it is, of course, prejudicial in the second. Hæmoptysis rarely occasions anxiety, and generally ceases without treatment, though in infarct the sputum may continue to be blood-stained for a week or more.

The milder degrees of cardiac discomfort are generally relieved by local applications, hot fomentations, blisters, anti-phlogistin, an ice poultice, etc., but if severe, require opium in some form. The pain of hepatic congestion is best treated by dry-cupping followed by hot poultices, or by leeching, the bowels being kept active by mercurials.

Gastro-intestinal symptoms sometimes entail anxiety, especially if vomiting is repeated and severe, and interferes with nutrition. Here, again, mercurials are most likely to be of use, and vomiting often lessens as soon as the bowels have been freely moved; a mustard poultice over the epigastrium sometimes helps. Whey, albumin water, and weak tea are most likely to be retained. The usual gastric sedatives are of little value. In severe repeated vomiting water is often well borne and may be retained, at any rate in part, even when all food is rejected. Even if it is returned, it flushes out the stomach and frequently makes the patient less uncomfortable.

4. The Action of Digitalis, as shown in healthy animals, is somewhat complicated, as, besides its local irritant action, it affects the central nervous system, the heart, and the arteries. It stimulates the vagus, slowing the rate of the heart and lessening its tone and constricting the arteries.¹

¹ Cushny, A. R., and others, *Heart*, 1912-13, iv., 33. Mackenzie, J., *Heart*, 1910-11, ii., 273.

It strengthens and prolongs ventricular systole by its direct action upon the heart, and increases its tone, so that relaxation is less complete; and it exalts its excitability. It produces constriction of the arteries directly as well as through the nervous system. The ultimate result, however, varies, as the nervous and cardiac mechanisms are in some ways opposed. With small doses the cardiac contractions become less frequent but more complete, so that the ventricular output is increased, and the arterial pressure is raised. With larger doses the inhibitory action is augmented, the contractions become less frequent and perhaps irregular, and, though diastolic relaxation becomes more ample, the ventricular output lessens. With still larger doses the excitability is so exalted that the pulse rate becomes very frequent, and the auricles may pass into fibrillation, while the ventricular action becomes irregular and the output is still further diminished. The irregularity may be due to several causes, vagus inhibition, auricular fibrillation or heart-block.

The crisp results obtained in experiment on normal hearts do not, however, convey as precise indications for the use of digitalis in actual practice as might be anticipated, as the hearts concerned are never normal, and in cardiac failure the whole metabolism of the patient is often out of gear. The uses of digitalis have to be determined by actual experience in sick people.

Withering's¹ remarks on the "constitution of patients" in his *Account of the Foxglove* are extremely interesting. "Digitalis," he says, "seldom succeeds in men of great natural strength, of tense fibre, of warm skin, of florid complexion, or in those with a tight and cordy pulse. If the belly in ascites be tense, hard, and circumscribed, or the limbs in anasarca solid and resisting, we have but little to hope. On the contrary, if the pulse be feeble and intermitting, the countenance pale, the lips livid, the skin cold, the swollen belly soft and fluctuating, or the anasarcaous limbs readily pitting under the pressure of the finger, we may expect the diuretic effects to follow in a kindly manner." Translated into current phraseology, Withering's remarks mean that digitalis is not likely to prove helpful in cases of dropsy with high arterial

¹ Withering, W., *An Account of the Foxglove*, Birmingham, 1785, 189

pressure, and that it is likely to be of value if auricular fibrillation has ensued.

The first point which should be determined in every case of cardiac failure is the height of the blood-pressure. A sphygmomanometer is not required, the finger alone can decide the point, for if the blood-pressure is high, eliminative treatment is required, and digitalis will fail unless it is accompanied or preceded by treatment of this kind. A hundred years after Withering wrote, William Murray¹ discussed the point as follows: "The administration of (mercury) in cardiac dropsy . . . is as old as the hills, and we old-fashioned physicians know well enough that thirty or forty years ago no one thought of treating those conditions except by a mercurial pill, followed by a saline or jalap purgative, and a diuretic mixture containing digitalis." The actual height of the blood-pressure is immaterial. Digitalis frequently acts well if the pressure is greatly above normal, if it is combined with treatment of the kind suggested by Murray; and, if otherwise justifiable, the administration of digitalis may be delayed with advantage for two or three days.

There is, of course, the risk that digitalis in these cases may produce such an elevation of the blood-pressure that a cerebral vessel ruptures. We are not aware of such an accident in our own experience, and we are at one with Sir James Mackenzie in his statement that medicinal doses comparatively rarely affect the blood-pressure to any appreciable degree. It is always wise to administer digitalis in a simple diuretic mixture—for example, in combination with citrate of potassium and liquor ammoniæ acetatis, or with iodide of potassium; or the well-known Guy's pill may be utilized.

The question is discussed in detail (p. 536) in connection with arterio-sclerosis, which, we would remind the reader, is very often associated with valvular disease of the heart. Of other drugs in these cases caffeine citrate and diuretin are probably the most useful. G. A. Gibson² recommended the combination of potassium citrate, caffeine citrate, and infusion of digitalis, which we have used with success.

¹ Murray, W., *Rough Notes on Remedies*, Lond., 1896.

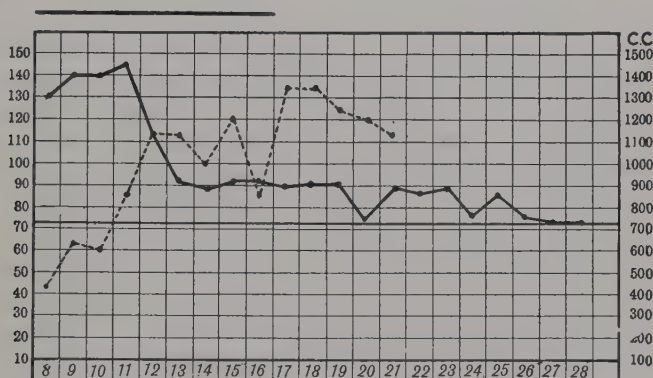
² Gibson, G. A., *Diseases of the Heart and Aorta*, Edin. and Lond., 1898.

The majority of writers state that digitalis is often useless in senile heart disease, and consider that this is due to fibroid or other disease of the cardiac muscle. The fact is, of course, well known ; but the failure of digitalis is more often due to associated arterio-sclerosis and imperfect elimination than to actual disease of the myocardium.

The Action of Digitalis in Auricular Fibrillation.—

The occurrence of a weak, irregular pulse in dropsy was stated by Withering to be an indication for the use of digitalis. Sir William Broadbent gave a more detailed description, stating

ŒDEMA



TR. DIGITALIS

7 30 90 15 30

FIG. 238.—AURICULAR FIBRILLATION. RESPONSE TO DIGITALIS.

The line represents the urinary output ; the — line the ventricular rate. Male aged fifty-one, syphilitic myocarditis.

that a *frequent* weak, irregular pulse, accompanied by œdema and a scanty, concentrated, turbid urine, were the special indications ; and Sir James Mackenzie, analyzing his results from the standpoint of the varying rhythms that may be met with in cardiac failure, states that it is most useful in auricular fibrillation, the pulse rate slowing concurrently with improvement, though the irregularity persists.

The slowing of the pulse rate in these cases is probably due to stimulation of the inhibitory mechanism and depression of the conductivity of the auriculo-ventricular bundle.

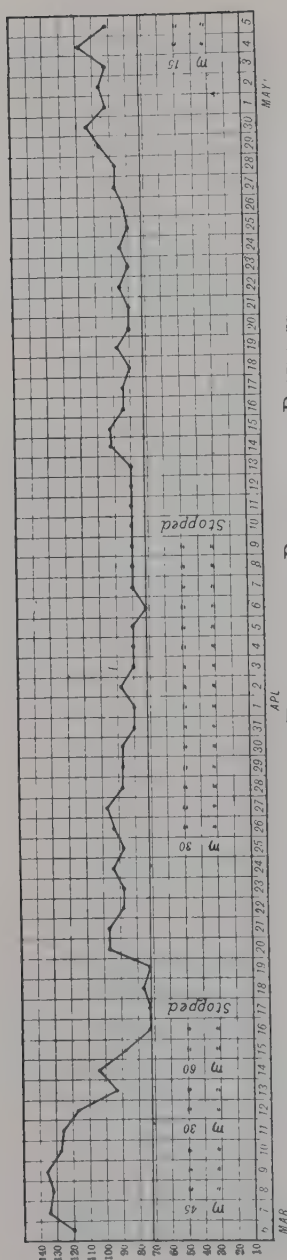


FIG. 233.—AURICULAR FIBRILLATION. RESPONSE TO DIGITALIS.

Chart showing the ventricular rate, which falls during the administration, and rises after the cessation, of the drug. Male, aged twenty-two. Mitral valvular disease.

Cushny's¹ conclusions, that the slowing of the ventricles is due to a direct action of the drug on the heart, are not in accordance with clinical experience.

Sir James Mackenzie recommends a daily dose of one drachm of the tincture, divided into three or four equal doses. French writers advocate isolated massive doses. But the repeated administration of the smaller doses seems to us to be preferable, as minor adverse symptoms can then quickly be checked by the withdrawal of the drug. In our experience smaller doses than a drachm may be sufficient, but the administration of 60–90 minims daily, at the commencement of treatment, has seemed to us to accelerate improvement, and the larger doses are sometimes alone efficacious. The effects of the drug are not noticeable for three or four days. In the majority of patients a good result is obtained when the pulse rate reaches normal figures, but some patients experience most benefit when it is still lower. Balfour stated that he had successfully kept the pulse running at 40 for days! Such cases are, however, exceptional.

¹ Cushny, A. R., *Proc. Roy. Soc. Med.*, 1912, v., Therap. and Pharm. Sect., 200.

When all symptoms have disappeared and the pulse rate is within normal limits, the dose should be discontinued and general tonics administered. But careful watch must still be paid to the pulse rate. In some patients it remains infrequent after digitalis has been stopped. In others, after a few days, usually in the second week, the pulse rate begins to rise, and coincidentally the symptoms recur (Fig. 239). *In these patients digitalis must be administered regularly, the optimum dose being ascertained by experiment.* One of our patients who was suffering

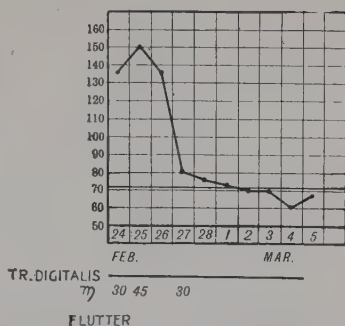


FIG. 240.—AURICULAR FLUTTER. RESPONSE TO DIGITALIS.

Chart showing ventricular rate. The rhythm was normal on the 27th February. Male, aged fifty-one. Chronic nephritis.

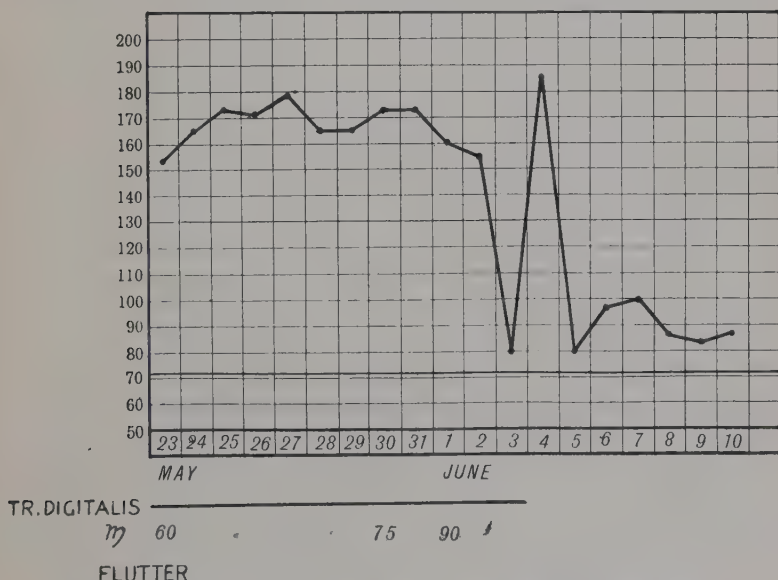


FIG. 241.—AURICULAR FLUTTER. RESPONSE TO DIGITALIS.

Chart showing ventricular rate. The auricles were fibrillating on the 3rd June, and fluttering on 4th June. On the 5th June and subsequent days the auricular action was normal. Male, aged sixty. Aortic valvular disease.

from mitral disease and fibrillation stated that she had taken digitalis regularly for seven years. After her admission into hospital improvement was rapid, and the drug was stopped. But as soon as she went to a convalescent home her symptoms recurred, though they were readily kept in abeyance by regular small daily doses. In such cases 10-20 minims of the tincture are generally sufficient.

We are unable to predict whether our patients who are suffering from fibrillation will require, or not, continuous administration of digitalis. The question can only be decided by careful clinical observation. But we have noticed that those patients who do not require regular doses are those who live on indefinitely though the fibrillation continues. One of our patients, who died at the age of seventy-five, had suffered from fibrillation for twenty-six years. Another, a medical colleague, who only relinquished practice after the last war, is in fair health at the age of eighty-two, although his auricles have been fibrillating for, he thinks, nearer forty than thirty years! In both patients the pulse rate was never frequent.

The Action of Digitalis in Auricular Flutter

In cases of auricular flutter the frequency of the ventricular contractions can generally be checked by digitalis, and the symptoms disappear as soon as the pulse rate returns to normal figures. Large doses, 60-90-120 minims of the tincture daily, are required. The dose should be intermitted as soon as the pulse rate becomes normal.

In most of these cases the normal rhythm returns more or less suddenly (Fig. 240), but this is not invariable. In some the flutter persists in the auricles though the ventricular rate lessens. In others the auricles pass into fibrillation (Fig. 241), and subsequently, in about half the cases, with cessation of the digitalis, the normal rhythm returns, at any rate temporarily. In several of our patients, however, the fibrillation persisted. This result, though unfortunate, is not necessarily dangerous, for in fibrillation the ventricular rate can as a rule be kept within normal limits. Flutter, if accompanied by a frequent pulse rate, invariably leads to grave cardiac failure within a few days. In one of our patients who has

been subject to the disorder for forty years, for many of which he paid little attention to his discomforts, grave symptoms now make their appearance if an attack lasts for more than an hour or two.

In flutter and in fibrillation danger lies in a frequent ventricular rate. If the ventricular rate is reasonable, the condition of the auricles is relatively immaterial.

Extrasystoles do not contra-indicate digitalis, for though they may make their appearance when the rate of the contractions lessens, their appearance is probably due rather to the prolongation of diastole than to increased excitability. When stimuli arise at frequent intervals, diastole may be too short to permit ectopic contractions to arise, and a coupled rhythm rarely occurs with a pulse rate of over 100, whether the auricles are in fibrillation or responding to sinus stimuli. We have, however, an electrocardiogram showing a ventricular extrasystole when the pulse numbered 232! (Fig. 172.) It occurred in a patient who was suffering from a paroxysmal attack of nodal rhythm.

Heart-block may be produced by digitalis in the clinic as in the laboratory. Sir James Mackenzie has utilized this reaction to prove the existence of those forms of partial block which are not associated with an intermittent pulse, the pulse becoming intermittent on the administration of digitalis as the block becomes exaggerated and occasional impulses fail to pass through to the ventricles. In exceptional cases, with a normal cervical tracing, block may also ensue, apparently from the existence of a latent weakness of conduction. The existence of a *partial* block is, thus, a contra-indication to the use of digitalis, as the block will be intensified. In *complete* block, however, this contra-indication disappears.

In patients with a *normal rhythm of the heart* the beneficial results of digitalis are rarely associated with such striking reductions of the pulse rate as are seen in fibrillation. The beneficial results in patients with a normal rhythm are probably due to an improvement of the *tone* of the heart, and a dilated heart often shrinks rapidly under the influence of the drug. The frequency of the pulse rate in fever, exophthalmic goitre, acute endocarditis, etc., is seldom influenced

to any marked degree, though other good results may be well shown. The value of digitalis in pneumonia is often distinct. A man, aged thirty-nine, was admitted into hospital on the fifth day of a pneumonia. He was slightly jaundiced and very prostrate, the pulse being soft and quick, and the second sound in the pulmonary area less emphatic than the first. Forty-eight hours later, coincidently with the administration of digitalis, the pulse was stronger and the wave better sustained, though the rate had not decreased from 120. The second pulmonic sound had now the emphasis. During the crisis on the eighth day the heart for a few hours showed signs of failure, but it quickly recovered and convalescence was good.

It may be necessary to administer considerable doses of digitalis and to push the drug until either the desired result is obtained or symptoms of intolerance appear. The old rule that nausea or vomiting, increased frequency of the pulse, or diminution of the output of urine, indicate withdrawal of the drug, is a useful guide, and dangerous symptoms will not occur if it is observed. The drug should also be intermitted if the pulse rate falls to 50, or if a coupled rhythm appears.

The dangers of digitalis are in all probability exaggerated. In myocardial disease sudden death is not uncommon, but its occurrence when the patient is taking digitalis is not necessarily the result of the drugging. Balfour quoted a case where death ensued after 211 grains of the powdered leaves had been taken in five weeks; but definite symptoms of intolerance had been present for four weeks prior to death. We have only seen one case in which sudden death ensued, and the drug might perhaps be blamed for the result. The patient, who was not under our care, was suffering from symptoms of cardiac weakness, which had followed the excessive use of beer. There was no valvular disease, but the heart was very large. Under full doses of digitalis the symptoms were relieved, the pulse rate falling to about 50. There were no other symptoms of intolerance, but he died suddenly one morning after getting out of bed.

The use of digitalis in cases of *aortic incompetence* has been much discussed. Theoretically, prolongation of diastole seems inadvisable in these cases, as it will increase the amount of

blood which returns into the ventricle. But, as has been already mentioned, failure in aortic cases is due to dilatation of the heart, and is often accompanied by mitral incompetence. The loss of tone which is present is an indication for the drug, and clinical experience has shown that much benefit may be derived from its use.

Many preparations of digitalis have been advocated, but the standardized tincture, the *fresh* infusion, the Nativelle granules, and Guy's pill,¹ are generally found to be effective. It is said that the infusion is particularly valuable in dropsy, and the tincture in fibrillation, while the pill is indicated in cases with defective elimination. Trousseau's wine² is often well borne by irritable stomachs. If nausea or vomiting contra-indicate oral administration digitalin may be given subcutaneously, but these symptoms may be produced by both oral and subcutaneous administration.

Of the other cardiac tonics strophanthus and squill have the best reputations. Strophanthus is said to have little action upon the arteries, and thus to be indicated in high blood-pressure. But it is alleged to be irritant to the kidneys, and so contra-indicated if they are diseased. In our experience strophanthus and digitalis are equally efficacious. In urgent cases strophanthin may be given by intravenous injection in doses of 0.5–1 mg. (about $\frac{1}{130}$ – $\frac{1}{60}$ gr.), or strophanthone (Park Davis) in doses of 0.5 c.c. of the dilute

¹ Guy's (Baillie's) pill.—Pulv. digitalis fol.; Pulv. scillæ; Pil. hydrarg.; ana gr. 1.

² Vin Trousseau.—White wine, g. 750; Squill bulbs, g. 5; Juniper berries, g. 50; Foxglove leaves, g. 10; macerate for four days. Add Potass. Acetat., g. 15; and filter. Dose, one teaspoonful.

The following prescriptions are useful:

Potass. citrat., gr. 20; Caffein. citrat., gr. 5; Infus. digitalis, drm. 4; Syr. Zingib., min. 20; Aquam, ad $\frac{3}{4}$ i. (G. A. Gibson.)

Potass. citrat., gr. 15; Liq. ammon. acetat., drm. 1; Tinct. digitalis, min. 5–15; Syr. tolut., min. 30; Aquam camphor., drm. 4.

Spir. ammon. aromat., min. 30; Spir. ætheris; Spir. camphor.; ana min. 15.

Potass. citrat., gr. 20; Tinct. strophanth., min. 10; Succ. scoparii, min. 60; Syr. Zingib., min. 20; Aquam, drm. 4.

Potass. iodid., gr. 5; Tinct. strophanth., min. 5–10; Syr. Zingib., min. 20; Aquam, drm. 4.

solution. Ouabine (Arnaud), another strophanthus preparation, may also be given intravenously or by the mouth.

Caffeine, theobromine (diuretin, theobromose), camphor, and convallaria are of use as adjuvants to the digitalis group, or in cases where a temporary cessation of such drugs is advisable. They are particularly valuable if the blood-pressure is high.

Alkaline diuretics, such as the citrates and acetates, should invariably be given with digitalis. They may be introduced into the mixture, or given separately in such forms as Imperial drink.

The diffusible stimulants, alcohol, ammonia, ether, are often useful. In cases of faintness they are the best immediate stimulant, whether inhaled or swallowed, and their action can be assisted by hot fomentations over the heart. If oxygen is being administered it can be bubbled through alcohol or ether, so as to obtain the effects of both. For continued oral administration alcohol is of more use than ammonia. Brandy and champagne seem the most valuable forms. Their value may be more on account of their vaso-dilator than their stimulant action.

In *syphilitic* cases specific treatment should invariably be given, in addition to that required by the condition of the heart. Not infrequently improvement ensues under the usual stimulant treatment but stops short of recovery, which, however, occurs when mercury and iodide are exhibited in addition. This is particularly the case in aortic valvular disease, which in our experience is *almost invariably the result of syphilis if there is no clear history of antecedent acute rheumatism*. The Wassermann test should be applied in doubtful cases, but a negative test should not necessarily contra-indicate specific treatment. One of the various arsenical preparations, novarsenobillon, neokharsivan, galyl, etc., may be given, but we are not inclined to believe in *therapia magna sterilisans* in these cases, and use moderate doses (g. 0.2-0.3 weekly) combined with courses of mercury and iodide. Liquor arsenici et hydrargyri iodidi is often useful, and is generally well borne.

The general nutrition of the patient must always be borne in mind, as cardiac failure may be due to general as well as

to cardiac causes. Anæmia, starvation, post-febrile debility, may all be accompanied by definite symptoms of cardiac insufficiency, which shows improvement only when the general, and the cardiac, nutrition improves. Arsenic, iron, strychnine, quinine, are thus all of value, particularly after the acute symptoms have subsided and convalescence has commenced.

CHAPTER XXII

ANGINA PECTORIS .

ANGINA pectoris is not a disease of the heart, but merely a single symptom, arising from many causes, which from its dramatic character dominates the stage. If there is anything that is certain about it, it is its uncertainty. An attack may occur once in a lifetime, or at intervals for years. The initial seizure may prove fatal, or recurring attacks may continue indefinitely without gravely interfering with the business of life. But in general, sooner or later, it eventually causes death unless the patient dies beforehand, so to speak, from some accidental infection or incident.

The striking feature of most of the diseases of the heart, even mortal disease, is the *absence* of pain. Anguish may be great from shortness of breath, a feeling of suffocation, or consciousness of the cardiac contractions, but actual pain is rare. In angina pain is the dominating and all-absorbing symptom, "a suffering as sharp as any that can be conceived in the nature of pain," and often accompanied by something that is beyond the nature of pain, a sense of impending death. The site of the pain is behind the sternum, often passing through the chest to the back, and it comes suddenly and goes suddenly. This may be the whole nature of an attack.

Angina pectoris varies greatly in its severity in different patients. Slight forms are extremely common; the grave forms correspondingly rare. In the milder degrees it is merely a mild uneasiness, "an indefinite sense of discomfort about the chest, with no distinct localization and no particular characteristics." There may be sensations of numbness or deadness, oppression, constriction or tightness, ranging in degree until they become actually painful. The pain may be dull or sharp, bearable though severe, or exquisite in its agony.

In a general way it is felt in the cardiac region, beneath the sternum or on either side, more commonly the left. As a rule, the character and the distribution of the pain in separate attacks in the same individual are constant. The pain may be confined to this area, or may radiate to the back of the chest close to the spine, or upwards into the neck or the shoulders. It may involve the arms, passing downwards as far as the elbows or the fingers, generally along the ulnar side. If this occurs the arms may become powerless for a time. The pain rarely involves the radial aspect. The left arm is more frequently affected than the right, but the latter may be involved, either separately or in conjunction with the other. In exceptional cases, thoracic pain may be absent, and the arms alone affected. The painful areas are often tender to the touch. Very rarely they are, following an attack, insensative.

With all this there is often another sensation, a faintness or sinking or feeling of impending death, separate and distinguishable from the pain. It is not invariably present, and may occur independently of the pain, the *angina sine dolore* of Gairdner. Its presence is sometimes appreciable to the onlooker, the patient's countenance being indicative of something more than pain, however terrible that may be. In the typical case the patient is still during an attack, holding on to some support, unable to speak or move or even breathe from the intensity of his distress, pale perhaps or covered with sweat or with a countenance unaltered save for the expression.

The initial attack is generally the result of physical exertion, sometimes of usual degree, sometimes beyond it. Several of our patients suffered for the first time when hurrying to catch a train, or on lifting an unusual weight. But occasionally it happens when the patient is at rest, perhaps sitting quietly after dinner on a chair, or in one case lying in his bed reading. As time passes and attacks recur, the amount of exertion required to induce them becomes lessened, and they may occur on even trivial provocation, associated, as in John Hunter's case, with mental emotion, or with digestive disturbances.

The character of the pulse during the attacks varies in

different patients. In the majority little change is noticed, save some, usually slight, increase in the rate. In some *the blood-pressure is raised*. In one of our patients it measured 160 mm. during the attacks and 130–140 mm. in the intervals. In other cases the pulse becomes *distinctly weaker*, and in one instance remained almost imperceptible for several minutes. The rhythm rarely alters during an attack, but we have observed all the recognized changes of rhythm in these patients.

The duration of attacks varies. The onset and cessation are generally abrupt, and the seizure lasts for a few seconds or a minute or two. It rarely lasts for ten minutes, but we have observed one persist for forty-five. Sometimes there is a rapid succession of attacks for several hours. If they are trivial the patient may feel quite well within a few minutes, but if they are severe he may be exhausted and tired for some hours or even days. One of our medical friends completed an operation upon which he was engaged during a slight seizure, though after another attack he was so exhausted that he had to stay in bed for several days. Another goes to bed as soon as possible surrounded by hot bottles, and falls asleep, awaking refreshed and well. If he does not go to bed he is unfit for two or three days.

The termination of an attack may be accompanied by eructations, or the passage of a large quantity of urine; but this seems to be exceptional.

Following an attack, the patient as a rule feels quite well, save for the remembrance of his agony.

Etiology.—Angina attacks individuals in every rank of life, but the brain-worker more often than the labourer. It affects men more frequently than women, and though Wild has recorded a fatal case at the age of twelve, it is rarely met with under thirty years of age, and is most common in the fifties and the sixties. It occurs in association with every variety of arterial and cardiac disease, in renal disease, and in persons whose hearts are apparently sound.

The *cause* of the pain has been much disputed (Huchard quotes 80 suggested causes), and even now is not fully understood. It seems obvious, however, that it is not the same in every case.

				Osler.	Personal.	
Sex :						
Males	..	..	..	256	43	299
Females	..	..	..	44	11	55
				300	54	354
Age :						
1-20	..	..	..	—	2	
21-30	..	..	..	9	5	
31-40	..	..	..	42	5	
41-50	..	..	..	60	6	
51-60	..	..	..	93	17	
61-70	..	..	..	72	18	
71-80	..	..	..	20	1	
81-90	..	..	..	4	—	
				300	54	

1. In a certain number of cases *the blood-pressure is elevated at the time of the attack*. The elevation may be temporary. In one of our patients who has a dilated heart, the blood-pressure during an attack measured 160 mm. Hg., and in the interval 120 mm. In another, who suffers from a rheumatic mitral lesion, the pressure during an attack was 160 mm., and in the intervals 130-140 mm.

In another group seizures occur during a *period of high blood-pressure*, and cease when the pressure falls. This was the case in a stoker, aged fifty, who caught cold and a week later began to suffer from attacks of angina of short duration, upon any exertion. After a few days he entered hospital and his attacks ceased. His blood-pressure, on entry 215 mm. Hg., fell steadily after admission, reaching 145 mm. on the fifth day, and ultimately 135 mm. Coincidentally the left heart, which on admission was slightly dilated, recovered its tone. He was again in hospital, three and six years later, on account of attacks of bronchitis and gastritis. The angina had not returned. His blood-pressure varied between 135 and 150 mm. Hg.

Angina occurs frequently in high-pressure cases. The blood-pressure, in the intervals, was high in 14 of 29 cases of which we have records. Unfortunately we have few records of the pressure during an attack. In several of these patients we

have been confident from digital examination of the pulse during an attack that the blood-pressure was temporarily increased. In other cases it seemed unchanged. It thus seems clear that in a proportion of cases, but not the majority, attacks are only present when the blood-pressure is definitely increased irrespective of its height between the paroxysms.

2. In another group all the evidence points to *weakness of the heart*. A woman, aged thirty, who had had the uterus removed six weeks previously, had her first attack of angina while sitting upon a chair. The attacks recurred frequently for several months, and then ceased. Three years subsequently they had not returned. Several of the attacks were carefully observed. One commenced suddenly while she was lying in bed in the early morning. She lay speechless on her back, very pale, breathing infrequently and lightly. Morphia was at once administered. Immediately the breathing became more frequent and more full. The pulse was now irregular, very weak, and almost imperceptible. The first sound was practically inaudible, the second was distinct. The blood-pressure was lower than usual. After five minutes the pain passed off, but the pulse remained weak and irregular, and only slowly regained volume and power. After forty-five minutes the pulse was regular and full, and her general condition as before the attack.

A healthy man of sixty-two years, who was very hard worked at the commencement of the recent war, had his first attack of angina while sitting quietly at home after dinner. The attacks recurred for six months and then ceased. Four years later he was in good health. During his attacks he was collapsed, the apex impulse becoming imperceptible, and the pulse weak, irregular and soft. The blood-pressure ran about 120 mm. Hg.

Electrocardiographic evidence of myocardial weakness may be distinct in these patients. We have seen T_{II} flattened or negative, the $Q-R$ interval prolonged, and the ventricular preponderance altered. With cessation of the attacks the cardiogram may return to normal.

3. In the exceptional case the cardio-vascular system is apparently normal. This is particularly the case when angina is associated with toxic causes, such as the excessive use of

tea, coffee, or tobacco. The last was apparently the cause of angina in a healthy well-built man who seemed to be absolutely sound. The attacks had first troubled him six years previously, while serving in the army, but they ceased and did not recur until three months before his admission to hospital. The pain was severe, purely diurnal, and not associated with exertion or indigestion. It was, he said, directly connected with tobacco, occurring only when he smoked cigarettes, and ceasing when he ceased the practice. On entry his pulse was infrequent, about 40 per minute, of sinus rhythm, and his hands were tremulous. After admission he stopped smoking and his difficulties rapidly passed away.

Pathology.—Pathological data are unfortunately scanty, and the records in many cases are incomplete. We can only add two cases. One patient was a man aged thirty-nine, who died during an attack. The first part of the aorta was slightly dilated, and showed a moderate grade of atheroma. The aortic cusps were thickened and shrunken, and the apex of the left ventricle was involved in a marked fibrosis. The coronary arteries were normal. In the second, a man aged sixty-one, the whole of the arterial system was extensively diseased, and the aorta showed widespread syphilitic scarring. The right coronary artery was normal in character, but the left was almost obliterated at its orifice, and its lumen a little lower down was blocked by large calcareous masses. Numerous small infarcts were present in the lower two-thirds of the left ventricular wall, associated with a terminal pericarditis. The valves were normal.

The most common finding is disease of the coronary arteries. They may be calcareous, as in Jenner's early cases, or obstructed in their course by endarteritis, or at their orifice by a patch of aortic sclerosis. Only one coronary artery was found in the heart of Dr. Arnold, who died during his first seizure. In Osler's series the coronary arteries were diseased in 13 cases and the aorta in 4, while in one the heart and aorta seemed normal, save that the latter was small.

The varying site of the pain in different cases, whether local or referred, makes it clear that the source of the pain must be sought in different places, arterial or cardiac, right-sided or

left-sided, as the case may be. For a common site of the lesion would not produce a varying site of the pain.

Sir Clifford Allbutt¹ has drawn attention to the frequent association of aortic disease and angina, and considers that the source of the pain in the majority of cases is aortic stress. The association of attacks with exertion and elevation of the blood-pressure, and the frequency of aortic disease upon *post-mortem* examination, are certainly suggestive. But the same facts can be interpreted otherwise as throwing strain upon the heart, and at the same time interfering with its nutrition by implication of the coronary arteries. In some cases the evidence obtained during the paroxysms points clearly to cardiac weakness; while in others it is shown as clearly that the work of the heart is increased. It is easy to show that both causes may be effective. A case recorded by L. Humphry was due to rupture of the aorta after severe physical exertion. A. W. Harrington has observed a case due to myocardial infarct. The patient, a man aged sixty-two, the subject of an old-standing mitral lesion, was suddenly seized with angina of severe type. No appreciable change in his condition was detected until the next day when widespread pericardial friction became audible. We have seen two cases of angina in which myocardial infarcts occurred. In both, however, widespread arterial disease coincided, and the nexus of events is consequently in doubt.

It seems clear that angina is due to the interaction of several causes. Patients with a systolic blood-pressure of over 250 mm. may never suffer from it, and those dying from the cardiac failure of anæmia may be equally free. In the first group, the height of the pressure predicates a fairly sound heart and arteries; in the second the blood-pressure is well below the normal. But patients with lesions in either heart or aorta may suffer from the operation of causes which in any way throw an unusual strain upon either. A "tender" aorta resents a blood-pressure even slightly above the normal, a "tender" myocardium is unable to react to even a trivial strain upon it, and distress ensues. The "tenderness" may be due to toxic or to organic causes. So that *any* interference with the nutrition, or direct irritation, of the sensory nerve endings in the

¹ Allbutt, Sir Clifford, *Diseases of the Arteries*, Lond., 1915.

heart or the aorta may produce the symptoms. The pain experienced in the legs in intermittent claudication upon exertion is in many ways comparable to that form of angina which is due to obstruction of the coronary arteries.

The referred pain owns a similar cause. The stimuli, wherever arising, are conveyed to the corresponding segment of the spinal cord, which also serves peripheral areas, and as the sensory and localizing powers of the surface of the body are enormously in excess of those of the viscera, by a psychical error the pain is referred to the periphery instead of to the organ actually affected.

Tenderness, when present, owns a similar cause. The impulses reaching the spinal cord produce an unstable equilibrium in the spinal segments concerned, so that stimuli of ordinary intensity arriving from the periphery produce an exaggerated impression.

In many of these patients a general hyperexcitability of the nervous system co-exists. The reflexes, for example, are over-active, and the mental state unstable. So that trivial stimuli produce an excessive response, and are felt in an exaggerated degree. This point must always be taken into consideration with regard to treatment.

Diagnosis.—We would follow Gibson, Osler and others in their refusal to distinguish extra-cardiac causes of substernal pain. Angina varies in its severity and in its duration, but even the mildest attacks are of the same nature as the most severe, and there is but a difference in degree between the discomfort of the poorly developed soldier marching with his pack and his rifle, and the terrifying seizures which occasion death. During the recent war, a common cause was the debility of imperfect convalescence from sickness or wounds, often increased by the general nervous strain of warfare. In civil practice, there is a group of cases with an unstable vaso-motor system, who are subject to pallor and coldness, or blueness and coldness, of the extremities. In others the ultimate cause is gastro-intestinal derangement, with offensive stools and excessive flatulence. In others the action of the endocrine organs is deranged; *but in all the heart or the aorta are exposed to stress and strain.*

Prognosis.—It has been said with truth that the only certain thing about angina is its uncertainty, and this is probably as true to-day as when it was first uttered. But, in a proportion of cases, where the underlying cause of the attacks can be determined, the prognosis can be estimated with fair accuracy by consideration of the prognosis of the underlying pathological condition. The cause may be temporarily or permanently in action, acute or chronic, progressive or stationary. The exciting factors may be trivial or great, preventable or not, and the circumstances of the patient may permit, or not permit, of such precautions and care as are likely to prevent recurrence of the attacks.

In young persons with healthy hearts, whose attacks are due to definite indiscretions in diet, tobacco, exercise or the like, the outlook is favourable if the error is corrected. In syphilitic cases specific treatment may check the progress of the lesions. Acute processes may progress or heal, while on the other hand the inevitable advance of degenerative lesions cannot be stayed. To the hospital patient leisure is rarely possible, though in private practice the exertions of business and the like can often be avoided without deprivation or distress.

Arnold died in his first attack. John Hunter lived for twenty years after he first began to suffer. The patient whose attacks were due to tobacco (p. 393) was permanently relieved by cessation of smoking. The woman whose angina followed an operation (p. 392) made a good recovery. A young man, whose attacks followed influenza, was cured by six months' rest. The business man whose illness was due to overwork (p. 392), remained free from symptoms for the next four years, during which he limited his activities to his capability.

Even in the degenerative group the outlook is not invariably gloomy. One of our patients had his first attack at the age of forty-eight, following severe exertion. With limitation of exercise the attacks ceased, recurring, however, ten years later, when he was using dumb-bells. A mild amount of exertion now always produced pain, which was intensified by influenza at the age of sixty-one. His limitations have steadily increased, but he still enjoys life at sixty-eight, though unable to take much exercise. Evidences of cardio-vascular degenera-

tion have been distinct throughout his illness. This patient, who had until his first seizure led a very active life, was able to appreciate the nature of his difficulties, and to adapt his life accordingly. The result has been good. In others, who from choice or circumstance have not been able to do so, the results have been unfortunate.

Death may ensue suddenly during a seizure. It has been suggested that this may be due to the occurrence of fibrillation of the ventricles, which in experiment is always immediately fatal. Such a termination has been witnessed by one of us. In other cases the attacks are accompanied or followed by signs of progressive cardiac failure. The failure and the pain are probably the result of the same pathological cause, and the association is of serious import.

In a general way cases which present definite evidence of high blood-pressure have a better outlook than those which show signs of cardiac weakness. The presence of œdema, orthopnoea, albuminuria, cyanosis, engorgement of the liver, associated with angina, is of evil omen. But, even in such patients, the extra strain of bronchitis or a bout of alcoholism may have been the last straw, and removal of the cause may permit the cessation of the seizures.

Treatment.—*During an attack* the immediate relief of the pain, by any means, is urgently required, for pain itself may occasion death. Those cases whose attacks are accompanied by elevation of the blood-pressure are almost invariably instantaneously relieved by inhalation of amyl nitrite. If this fails, morphia or chloroform should at once be administered. The patients whose blood-pressure is not elevated during attacks are seldom relieved by amyl. Unfortunately attacks are rarely observed by the medical attendant, and the condition of the pulse may be consequently in doubt. Some help may be found in observations of laymen with regard to the condition of the pulse, pallor or flushing of the face, pallor or coldness of the extremities, etc., but in many cases the only test of the efficacy of the nitrites is experiment, and the judgment of the patient is decisive. If it is useless for the relief of pain it may be discarded.

In cases where the blood-pressure at the time of the attack

is not elevated, morphia may be required. If there is evidence of cardiac weakness, alcohol, ether, ammonia, and hot fomentations to the præcordia are often helpful, but the opiate is generally essential. A carminative liqueur such as kummel or peppermint may help, particularly if there is any distension of the stomach.

The treatment *between attacks* must be guided by the causal factors, and every effort should be made to elicit their exact nature. If the blood-pressure is high an attempt should be made to lower it, though not necessarily to normal limits if, as so frequently is the case, structural changes are already present in the heart and arteries, but to such a point as will lessen the strain upon the heart and the arteries. For, even in cases with definite renal cirrhosis, the high blood-pressure may be increased by temporary toxic causes, the removal of which may enable the organs to do their work without distress. The nitrites help, but the essential point is to remove the causes of the temporary increase of the blood-pressure.

These are numerous. One of our medical friends tells us that the cause of his attacks is gastro-intestinal, and the cure blue pill and salts. In another patient the stools at the time of attacks are always extremely offensive. Another finds salvation in limitation of his diet, another in stopping alcohol and tobacco. A common source is excitement and worry. Too often this is due to social or business causes, which cannot be entirely removed, but they can often be lessened.

Most frequently the cause is *physical exertion*. Our surgical colleagues, who have to treat a broken leg, always excite our envy, for there is no difficulty in persuading *their* patients of the need for rest. In angina the symptoms, though clamant at the time of the attacks, are absent in the intervals, and the patients in consequence find it difficult to realize that their diet, exercise, rest, etc., at a time when they feel in perfect health, still require careful regulation. In many cases rest, more or less absolute, is urgently required, and that not for a few days but for weeks and even for months. This is especially the case in those patients whose attacks are the sequel to an acute process, whether in the heart or the aorta.

In patients with definite organic lesions the occurrence of angina should be looked upon as comparable to the occurrence

of œdema, or breathlessness upon exertion. In a general way a similar line of treatment is indicated, such as that discussed in detail on pp. 370-372.

Iodide of potassium, caffeine, and the bromides are sometimes useful. The former probably owes its reputation to cases with syphilitic lesions, and in our hands has been useless in non-specific cases. The bromides are useful in nearly every case, particularly in those patients who are hypersensitive and sleep badly. Caffeine is of course a cardiac stimulant. The activity of the various nitrites varies with the different preparations. Amyl nitrite acts almost instantaneously, but its effects are of short duration. Sodium nitrite and nitroglycerine are less rapid in their action, but their effects are of longer duration. Erythrol tetranitrate has the most prolonged action, but even its effects pass off within two or three hours. The dosage of these drugs must be regulated accordingly. They are of particular value in an emergency, and they cannot be given over long periods with advantage or safety. In at least two of our patients a nitrite habit has been established, with detriment to the general health.

The general tonics, such as arsenic, quinine, strychnine, and iron, may all be of value in particular cases, if the general health is impaired.

CHAPTER XXIII

DISORDERED ACTION OF THE HEART

THE disability known as "soldier's heart," the "irritable heart of soldiers," and "disordered action of the heart," the latter being the official nomenclature, has recently received considerable attention, as it was peculiarly frequent among the troops, of all nationalities, during the recent war. It is, however, in no way a specific disease, but merely a collection of symptoms of varied origin, some of which are referable to the heart.

The Symptoms.—The majority of these patients complain of *pain* in the region of the heart. It is generally referred to the apex and confined to the cardiac area ; but occasionally radiates into the arm or into the back. It is usually induced by exertion and absent during rest, but it may follow mental emotion, or occur during the night. The majority, too, complain of *shortness of breath*. This, like the pain, is generally related to exertion, which need not be extreme ; but occasionally it is nocturnal in onset, or associated with signs of mental distress. Some complain of *giddiness*, sometimes on exertion, but more commonly on sudden changes in position, such as rising from the recumbent position. Some complain of *palpitation*, which generally occurs in paroxysms. These are often only induced by exertion, but they may be nocturnal in onset, or associated with signs of mental excitement or strain ; or, but rarely, occur irregularly, without obvious cause. A few complain of *faints*, in some of which consciousness is lost. Almost all these patients complain of *exhaustion* and tremors, and many of *headaches* and *sleeplessness*.

Etiology.—In a certain number of these patients routine examination reveals definite physical disabilities. In some the

heart is not directly involved (Hume,¹ 8·3 per cent. ; Ritchie,² 12·4 per cent.), the patients suffering from phthisis, emphysema, exophthalmic goitre, etc. In others there are gross cardiac or arterial lesions (Hume, 5·5 per cent. ; Ritchie, 14·6 per cent.). But in the large majority of cases (Hume, 86·2 per cent. ; Ritchie, 73·0 per cent.), *the most careful examination fails to reveal any objective cause in the cardio-vascular apparatus.*

Investigations have been made by many observers in the hope of discovering some common cause of the disability. The early papers are chiefly concerned with the dress and equipment of the troops, which were, of course, notoriously defective in the crusades of Saint Louis, both men and horses wearing armour in the month of April at the landing at Damietta ! In the sixties they were still defective, but the changes introduced at the end of last century produced a kit to which exception can hardly be taken.

The investigations³ which were conducted at home during the recent war have on the whole failed to reveal any new etiological data of importance. Cigarette-smoking has been exonerated. The vital capacity and the alkalinity of the blood are not at fault. The intrinsic action of the heart is normal. The rise in blood-pressure and in pulse rate produced by exertion are similar to those in healthy men. But the hearts of these patients are somewhat smaller than normal, and the cardiac action is unstable, and unduly affected by causes (emotion, exercise, changes in position) which in health produce little alteration.

The causes of "D.A.H." are numerous and vary considerably in different campaigns, and at different periods. At the commencement of the recent war, the physical standards were high, and a breakdown resulted as a rule from the activity of some definite *extrinsic* agency. In 1917 the physical standards were lowered, and breakdowns were more frequently due to an *intrinsic* weakness in the physique of the patient. The causes of the strain varied greatly in the different war areas, which comprised tropical, sub-tropical, temperate and arctic climates.

¹ Hume, W. E., *Lancet*, 1918, i., 529.

² Ritchie, W. T., and others, *Lancet*, 1920, i., 852.

³ *Heart*, 1917, 1918, 1919.

The *old Army* was composed of volunteers, who entered in early life before growth was complete. A small proportion of these men proved incapable of completing their training, on account of the development of such symptoms. The new Armies contained a large number of men, between twenty-five and forty years of age, whose growth at the time of their entrance was completed, and a larger proportion proved incapable. Many of these were men who, from necessity or choice, had engaged in sedentary occupations, and had had little opportunity of indulging in physical exercises. Fully grown and "set" at the time of their entrance, there was little possibility of much physical development, however carefully their training might be graded. Many, indeed, became fit for exertions far beyond their former powers, but many developed "D.A.H." at an early age. In others the training was well borne, but the stress of the campaign readily produced distress. There is little physical resilience after forty years of age, and even in the thirties, and at these ages an isolated physical overstrain, which in the twenties would be rapidly overcome, often leads to permanent disability. The old soldier, nearing forty years, if once incapacitated, rarely recovers his former vigour. In some instances the strain is severe and of short duration. In others it is of less intensity, but prolonged. The results, however, are similar.

The prolongation of the war gave origin to many new problems. To most men many years of campaigning are impossible, and they become exhausted and war-weary. The breakdown may be physical or mental. In the former case any organ may be involved, and as "heart disease" to the layman spells sudden death, any cardiac discomfort receives an attention which would not be accorded to a similar discomfort in the systemic muscles.

Hume's statistics have shown that some of these considerations are correct. The majority (58·8 per cent.) of his patients had been engaged in the lighter forms of occupation, and the majority (54·2 per cent.) had suffered from similar symptoms in civil life; 5·6 per cent. dated the onset from some severe exertion.

In a small group of cases the symptoms, though referable to the heart, are evidently of nervous rather than of cardiac

origin (Hume, 5·7 per cent. ; Ritchie, 4·8 per cent.). There is only one "great" war for each of us, the *first*—that war in which we first see a man killed, and realize that at any moment our hour may come. Later wars are merely repetitions ; heat and cold, thirst and wet, dust and mud, boredom and excitement, exhilaration and fear ; a combination which in many men produces symptoms referable to the heart.

In some of these patients other nervous phenomena (convulsions, chorea, etc.) have been manifested in early life, or co-exist. In some, the symptoms have succeeded a gross nervous disturbance (shell-shock, entombment), or the prolonged nervous strain of modern warfare. In others some family or business anxiety has occurred. These patients are "nervous," tremulous, and excitable, and their reflexes are over-active. They are hyper-sensitive, and intolerant of peripheral impressions, which are recognized by the normal individual to be of natural origin and trivial significance. In some the disability has been largely caused or fostered by their medical officer. In one case, for example, a diagnosis of aortic aneurysm made by an enthusiastic radiographer permanently incapacitated a healthy man. But neurasthenia is by no means a common cause of "D.A.H.," though its existence exaggerates any symptoms that may be present.

The efficiency of any effort, mental or physical, depends for its success upon a satisfactory supply of blood to the brain and the tissues.¹ The venous "lakes" in the abdomen, lungs, and skin, are each more than sufficient to contain the whole of the blood ; and if the effort is to be successful, these lakes must be depleted and their blood made available for the arterial supply. If the abdomen is flabby and pendulous, if the lungs are emphysematous, if the costo-vertebral articulations are fixed, the venous reservoirs cannot be emptied, and the arterial output will be insufficient. In these patients the cervical veins become swollen when the recumbent position is assumed : and still further distended if pressure is exercised upon the abdomen. The mechanism for the evacuation of the "lakes" in normal individuals is partly cardiac and partly extra-cardiac, and under the control of the nervous system. The

¹ See Wilson, R. M., *The Nervous Heart*, Lond., 1919 ; *The Hearts of Man*, Lond., 1918.

control is exercised by the vagus and the sympathetic nerves, the action of the two being antagonistic, and one or other dominant from time to time according to the needs of the organism. If the action of one or other is inefficient, or the balance between the two irregular, the result will not be satisfactory, and the effort will be ineffective. In some of these patients the nervous balance can be shown to be irregular. The vagus action may be excessive, the pulse becoming less frequent and the blood-pressure falling when a deep breath is taken and held, a reaction which is abolished by atropine. The sympathetic action may be excessive, the reaction to adrenalin being abnormally great. These hyper-irritable nerves react actively but irregularly to stimuli of minimal character, and symptoms of distress ensue.

In hot climates vaso-motor disturbances are not uncommon. Many men show œdema in the feet at night without other obvious signs of visceral disturbance; and the œdema disappears when the cold weather comes. Every one, too, knows the feeling of limpness and inability to perform the slightest physical exertion which obtains on hot, sticky days, and the profuse sweating which occurs during exercise. The blood-pressure at these times is below the normal. After a rest, or with the cool of the evening, relief comes, sometimes almost instantaneously.

In a large group of cases the onset of symptoms is ascribed by the patient to some acute infection (Hume, 22.0 per cent.; Ritchie, 41.4 per cent.; Venning,¹ 20.22 per cent.).

One is apt to forget that a febrile illness, even though due to general causes (enterica, influenza), or disease of an organ other than the heart (pneumonia, dysentery), invariably produces recognizable pathological changes in the cardiac muscle. Fatty and granular changes are the rule, hyaline changes are common, and inflammatory reactions, as a rule of microscopic size but often numerous, are not infrequent, quite apart from microscopic lesions of embolic origin. The two former disappear as health returns, but the latter leave scars, which, though minute, may from their ubiquity interfere gravely with the cardiac strength. It is generally recognized that such an illness as enterica requires *prolonged* convalescence before the

¹ Venning, J. A., *Brit. Med. Journ.*, 1919, ii., 337.

soldier is able to resume the active duties of a soldier. A few weeks at a convalescent home are not sufficient. And in many cases even an apparently trivial illness may hit the heart heavily, especially if the patients "carry on" throughout the acute stage of the disease, exposing the weakened heart to the stress and strain of physical exertion.

In France, "P.U.O." pneumonia and rheumatic fever were the diseases which most frequently preceded D.A.H. In Egypt, diphtheria, rheumatic fever, and dysentery.

K. H. Reed and C. Walker¹ made some valuable investigations into the cardiac condition of men who were convalescent from these diseases, at Montazah in 1918. The patients admitted to this hospital were as a rule particularly debilitated, or had been seriously ill during the acute stages of their illness. One hundred and fifty-seven men who had suffered from dysentery were examined. In 123 the pulse rate at rest was above 100, and only 6 showed no evidences of cardiac insufficiency. Forty-six men had suffered from diphtheria. In 36 the pulse rate at rest was above 100, and only 2 showed no evidences of cardiac insufficiency.

R. H. Strong² also investigated the question. He found that 72.2 per cent. of 200 men, who had diphtheria three or four weeks previously, had still a pulse rate at rest above 90. He saw 95 of these men two months later, and 52.2 per cent. still showed this frequent pulse. Systolic murmurs were not infrequently present. Extrasystoles were sometimes present, and heart-block was also noted. The heart was sometimes dilated, as a rule during the acute illness, but also sometimes late in convalescence. In one case the condition was recognized in May, the diphtheria having occurred in January. The knee jerks were still absent.

In diphtheria cardiac disturbances may be due to myocarditis, the common cause, or to neuritis of the cardiac nerves. This latter is uncommon in other infections, but as has been already mentioned, myocarditis is the rule, in greater or less degree, in *all* infections if the illness is severe. There is no difference in this respect between civil and military practice. But in the former the opportunities for prolonged convalescence are usually greater than in the latter, and the physical strain in

¹ Unpublished.

² Unpublished.

civil employment is less severe. But every one has met with patients, especially after influenza, whose cardiac convalescence was tedious and imperfect, and who required to change their occupation in consequence. In civil life the weakling accepts his disability, and lives accordingly. In military practice an attempt is always made to restore a man to his former fitness.

In the majority of these patients the ultimate progress is good. With time and carefully graded exercise recovery is complete. Convalescence has merely been *delayed*.

The group of patients labelled "D.A.H." in Army parlance thus comprises disabilities of very varied origin. In some, organic disease, of cardiac or other nature, is present; in some the condition is largely a neurosis; in others it is merely imperfect convalescence or a form of physical weakness. True D.A.H. is by no means a common affection, and can readily be recognized upon examination. The nomenclature is correct. D.A.H. is *disordered action of the heart*.

A few examples will suffice. An orderly in the surgical theatre fainted one morning when on duty. He was a "B" man, who had worked hard for three years and had taken little holiday, for he disliked being absent at emergency operations. His heart was of normal size, and the sounds were pure, but of poor quality. His pulse was frequent, and notably irregular from extrasystoles. After walking quietly upstairs he was short of breath, and remained so for at least three minutes.

An officer had not been out of Egypt for five years. One morning he took some salts, which failed to act, had his breakfast and walked into town. Half an hour later he was found lying unconscious in the street, very pale and almost pulseless. He recovered consciousness in half an hour. A couple of hours later he was seen lying in bed. His colour was good and his pulse of fair value, but notably irregular from auricular flutter. Four hours later the rhythm suddenly changed, and the pulse became regular and its quality improved. Convalescence was uninterrupted.

An officer went for a ride one morning, and after breakfast suddenly fainted. In a few moments he had apparently recovered completely. The organs seemed sound, but his

blood-pressure was 20–30 mm. below his normal, œdema was constantly present in his feet, and he sweated on trivial exertion, while his pulse rate ran up to over 100. With the advent of cold weather his disabilities disappeared. The cause was evidently a vaso-motor disturbance.

In these patients evidence of organic cardiac disease may or may not be found. The term D.A.H. should be reserved for the latter group, the former being designated mitral stenosis, dilatation of the heart, etc., as the case may be.

CHAPTER XXIV

ACUTE PERICARDITIS

PERICARDITIS is less common than endocarditis, and only occurred in 158 of 3,842 patients who were admitted to hospital on account of heart disease ;¹ but its high mortality (63 of the 158 patients died during their residence) makes it an important cause of death in cardiac diseases.

The Lesions.—Pericarditis may be local or diffuse, and may be acute or chronic. It may be due to many micro-organisms, the pyogenic cocci, the pneumococcus, and the organism of acute rheumatism being those which are most frequently causal ; while a tubercular infection is by no means uncommon, both as a major and as a terminal event. In the acute stage the exudate may be sero-fibrinous, purulent, or hæmorrhagic. If recovery ensues, resolution may be complete ; but it is usually imperfect, and organization of the exudate occurs, with adhesion, partial or universal, of the visceral and parietal layers.

In the earliest stage the serous surface is hyperæmic, dull, and opaque, and minute hæmorrhages may be apparent in the vicinity of the vessels, with here and there a few flakes of fibrin. In the majority of cases, however, a fibrinous or fluid exudate is found on examination. The fibrinous exudate varies in character in different cases. It may be scanty and slight, and situated mainly around the great vessels ; or thin and more diffuse, covering the greater part of the heart wall ; or abundant and universal, involving both the parietal and the visceral layers. The deposit is usually thickest in the

¹ Lockhart Gillespie, *Edin. Hospital Reports*, 1898, v., 293.

Andrew, J. G., *Age Incidence, Sex, and Comparative Frequency in Disease*, Lond., 1909, 156, 157.

vicinity of the coronary vessels. From the constant movement of the heart the surface is rarely smooth, generally presenting a "rippled," honeycomb, or shaggy appearance, with bands of varying thickness here and there uniting the two layers. In exceptional cases little fluid is effused, but as a rule the sac contains a few ounces, even in those cases in which the fibrinous deposit is most extensive and thick. In other cases the fluid effusion is considerable, and may amount to several pints, and the fibrinous exudate is relatively scanty. The fluid is yellowish-green in colour, and generally fairly clear, but it may be turbid. The specific gravity is about 1.018, much albumin is present, and clotting takes place if the fluid is allowed to stand. In severe infections the fluid may be frankly purulent, and it is occasionally hæmorrhagic in character. In the slighter infections the exudate may be completely absorbed, but in the majority of cases adhesions form between the two surfaces, and are ultimately organized. In some cases calcareous material is deposited between the two layers (Fig. 242).

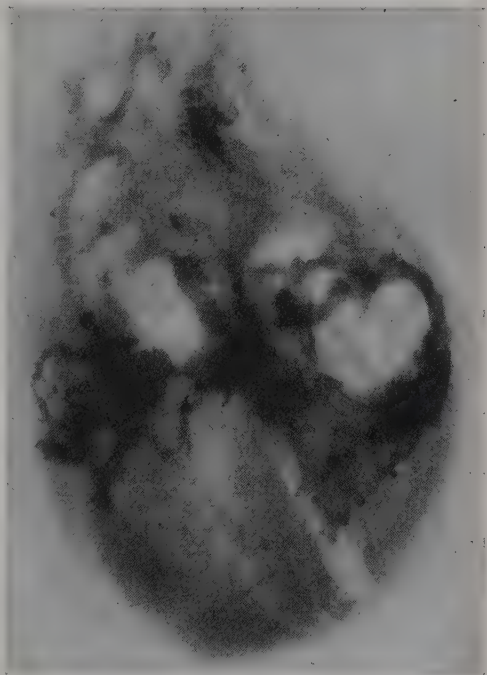


FIG. 242.—RADIOGRAM OF HEART SHOWING CALCAREOUS MATERIAL DEPOSITED BETWEEN THE LAYERS OF THE PERICARDIUM (K.M.C.).

In most cases the chief deposit occurs on the visceral pericardium, and the pericardial sac is but slightly involved; but in others the parietal layer is primarily or chiefly involved, and in this case the inflammation may extend outwards into

the mediastinum, and not infrequently involves the pleural sacs as well. In a few cases the primary focus is outside the sac, and a comparable lesion occurs, but with a different sequence. Tubercular pericarditis sometimes arises in this way from infected mediastinal glands.

The exudate in these cases may be fibrinous, with little tubercles scattered here and there throughout it. More rarely the tubercular masses are of considerable size, and they may even encircle the heart in a layer of some thickness. The fluid exudate is usually serous, but may be definitely bloody in character.

In acute pericarditis the superficial muscle cells may be seriously damaged, showing distinct fatty or other changes; but the connective tissue of the heart is little altered. The fibrous tissue around the main vascular trunks or the superficial layers of muscle cells may be more bulky than normal, and more thickly nucleated; but even this is rare, and in any case the lesion is always superficial. In adherent pericardium a similar state of matters is the rule, and we have only twice seen any considerable degree of fibrosis in the heart muscle. In both cases it was patchy in distribution, and in one case there was, in addition, widespread valvular disease. Myocardial lesions, however, are probably always related to causes other than the pericarditis. Acute endocarditis is usually accompanied by a varying amount of myocarditis, and not infrequently by pericarditis as well; and a myocardial infarct or abscess, if it approaches the surface, is necessarily associated with pericardial changes; but these are the result, and not the cause, of the myocardial lesion. In tubercular pericarditis, the superficial muscular layers alone are usually involved.

In mural endocarditis, on the other hand, the muscle is always deeply involved, as the infection spreads outwards along the lymphatic channels. In pericarditis the inflammation does not travel far against the lymphatic stream.

The "*milk spots*" which are so often seen on the surface of the heart are always superficial, and never invade the muscle. It seems probable that they are rarely the result of local acute pericarditis. They are most common in elderly persons, and are rare in childhood. They are generally situated on the front of the heart on the parts which are directly related to

the sternum and ribs, and are unusual on the parts which are covered by the lungs. They are probably due to mechanical irritation produced by the movements of the heart against the unyielding bony thorax.

The *functions* of the pericardial sac, when it is inflamed, are frequently gravely injured. In the acute stages it is infiltrated and thickened by inflammatory exudate, and its strength and resilience are seriously affected if at this stage it is distended by effusion, and are still more disturbed if the inflammation spreads outwards into the mediastinal structures, and adhesions form between the pericardium and the thorax or the neighbouring viscera.

The adhesions which follow acute pericarditis vary considerably in distribution and in site. They may be delicate, tenuous, and long, or dense and thick, and then usually short; and while they are as a rule, even when considerable, more or less localized, they may, in exceptional cases, be universal, and obliterate the pericardial cavity. Complete obliteration of the sac is unusual, but it is often divided into several compartments.

Adhesions may be present anywhere, and may interfere with the proper action of the heart, as they may embrace and strangle the *venæ cavæ* and the right auricle, or the pulmonary veins and the left auricle; or involve the aorta, the pulmonary artery, or the ventricles.

If the inflammation spreads outside the sac, the pericardium may become fixed to neighbouring structures, the sternum and adjacent ribs in front, the diaphragm below, the lungs on either side, or the vertebral column behind.

The Etiology of Acute Pericarditis.—Pericarditis may be associated with many diseases, and is rare as a primary event. It occurs in (1) the infections generally, in particular acute rheumatism; (2) intrathoracic disease of various kinds; (3) general and local sepsis; and (4) chronic disease, as a terminal event.

1. Of all the infections *acute rheumatism* is that with which pericarditis is most frequently associated, and its incidence in this disease is considerable (16 per cent.) though decidedly less than that of endocarditis (53 per cent.), by which, however,

it is commonly accompanied. It is almost equally frequent in the two sexes, and, like endocarditis, is much more common in early life, being present in 94 out of 100 cases of fatal heart disease observed by Sturges¹ at Great Ormond Street Hospital. It is generally considered to be most usual in first attacks of rheumatism, but in Church's series, 56 per cent. occurred in second or subsequent attacks. Its incidence is greater the more severe the arthritis, and it rarely obtains in those trivial rheumatic illnesses with which endocarditis, on the other hand, is so often associated.²

Roberts stated that it is not uncommon between the third and the sixth day of the arthritis. Balfour thought that the majority of cases occurred within the first week; and in

¹ Sturges, *Lancet*, 1894, i., 583, 653, 723.

² THE INCIDENCE OF PERICARDITIS IN ACUTE RHEUMATISM (GARROD).

					Acute Rheumatism.	Pericarditis.
					Cases.	Cases.
Latham ³	..	..	..	..	136	18
Peacock ⁴	..	..	..	..	233	36
Sibson ⁵ ..	..	..	..	..	326	63
Pye-Smith ⁶	..	..	..	..	400	96
Whipham ⁷	..	..	..	..	655	111
Osler ⁸ ..	..	..	..	..	330	20
Totals ..	..	..	..	..	2,080	344

Church: Of 155 cases of pericarditis occurring in acute rheumatism, 67 accompanied the first attack.

					Male.	Female.	
Church ⁹	..	..	..	..	12.22%	10.53%	2,290 cases
Lockhart Gillespie	..	..	..	..	68	51	
Sibson ..	..	..	..	..	35	28	

³ Latham, quoted by Garrod, A. E., *A Treatise on Rheumatism and Rheumatoid Arthritis*, Lond., 1890, 100.

⁴ Peacock, *ibid.*, 100.

⁵ Sibson, F., *Reynolds's System of Medicine*, Lond., 1877, iv., 186.

⁶ Pye-Smith, P. H., quoted by Garrod, A. E., 101.

⁷ Whipham, *ibid.*, 102.

⁸ Osler and McCrae, *The Principles and Practice of Medicine*, N.Y. and Lond., 1912, 760.

⁹ Allbutt and Rolleston's *System of Medicine*, Lond., 1906, ii., part i., 622.

Sibson's series¹ the signs were recognized within eleven days in nearly half the cases (30 out of 63 cases). But in exceptional instances the onset may be delayed indefinitely, as late as the sixty-first day (Sibson), or may supervene during a relapse. A married woman, aged twenty-seven, was admitted to hospital suffering from acute rheumatism on February 23rd. The arthritis soon subsided, but on March 20th a consolidation appeared at the base of the left lung. In the early part of April crops of subcutaneous nodules appeared, and on the 29th, two months after the subsidence of the arthritis, pericarditis was recognized. A girl, aged fifteen, suffered from acute rheumatism in December, 1910, chorea in April and June, 1911, acute rheumatism in November, 1911, and slight arthritis for a few days in January and April, 1912. With the last attack a pericardial effusion developed. In both of these patients acute endocarditis co-existed, and in the latter case the cardiac infection seemed to be persistent and responsible both for the recurring attacks of arthritis and the pericardial infection.

Chorea is responsible for many cases of pericarditis. In Sibson's series it occurred in 15 out of 21 cases of acute rheumatism which were associated with chorea. Osler found that it was present in 19 of 73 cases of chorea examined *post-mortem*; and Cheadle² has drawn attention to its association with the

¹ Sibson, F., *Reynolds's System of Medicine*, Lond., 1877, iv., 186.

² Cheadle, W. B., *Occasional Lectures on the Practice of Medicine*, Lond., 1900, 277-84.

THE INCIDENCE OF PERICARDITIS IN INFECTIVE DISEASES.

	Cases.	Pericarditis.	Per Cent.
Pneumonia (Musser and Norris) ³ ..	40,773	499	1.2
<i>Post-mortem</i> series (Musser and Norris) ³	2,128	268	12.6
Scarlatina (McCollum) ⁴	2,000	13	0.65
Enteric fever (McCrae) ⁵	1,500	3	0.2
Phthisis (<i>post-mortem</i> series) (Norris) ⁶	1,780	81	4.6

³ Musser, J. H., and Norris, G. W., Osler and McCrae's *System of Medicine*, Lond., 1907, ii., 610.

⁴ McCollum, J. H., *ibid.*, 352.

⁵ McCrae, T., *ibid.*, 145.

⁶ Norris, G. W., *Studies in Cardiac Pathology*, Phil. and Lond., 1911, 113.

other rheumatic manifestations, erythema, subcutaneous nodules, and tonsillitis.

Pericarditis is of rare occurrence in the other infections. It is most common in pneumonia (1.2 per cent.), scarlatina (0.65 per cent.), and enteric fever (0.2 per cent.), and may occasionally obtain in smallpox, measles, influenza, diphtheria, and gonorrhœa. In smallpox it is probably due as a rule to a secondary infection.¹

2. The incidence of pericarditis in *thoracic disease* is chiefly due to its association with pneumonia, pleurisy, and phthisis. Its occurrence in pneumonia is of serious significance, as its *post-mortem* incidence (12.6 per cent.) is much greater than that reported from the wards (1.2 per cent.). It is uncommon in pleurisy, save in empyema. In phthisis it was found *post-mortem* in 4.6 per cent. Some of these cases were not tuberculous, but due to a secondary pyogenic infection.

It may follow inflammatory lesions in the sternum, ribs or mediastinal glands, whether tubercular or pyogenic; inflammatory processes beneath the diaphragm, such as a sub-diaphragmatic abscess, or fractures of the ribs and penetrating wounds of the thorax. It may follow injury to the chest wall without fracture. This occurred in a man, aged eighteen, who dated the commencement of his illness to a blow received while playing at football, his chest being hurt by contact with an opponent's elbow. The pain at the time was not severe, but it became exaggerated next day and accompanied by shortness of breath. No other cause could be discovered.

3. Pericarditis is sometimes a terminal event in *pyæmic* cases. It may be secondary to a myocardial abscess, or occur apart from cardiac disease, as the result of a direct infection from the blood stream.

4. Pericarditis is not uncommon as a *terminal event* in chronic diseases of various kinds, the inflammation occurring on account of the lessened resistance to infection. It has been met with in anæmia, cancer, gout, and renal disease, and is

¹ *Diseases of the Heart and Aorta*, Philadelphia, 1910.

most common in the latter, in particular in renal cirrhosis,¹ while it is comparatively rare in acute nephritis. The micro-organisms concerned are chiefly the pyogenic cocci, but a tubercular infection is by no means rare.

The actual incidence of these factors varies in different series, mainly on account of differences in the class of case under observation; but the most common accompaniment is endocarditis, usually of rheumatic origin. The inflammation in these cases is presumably derived from an infected embolus involving a superficial vessel. In one of our patients the process was demonstrated macroscopically. The mitral valve showed a chronic endocarditis, the result of an acute rheumatic attack four years before, with a few small granulations of recent development superimposed. The artery in the posterior inter-ventricular sulcus was involved, about an inch from the apex, in an acute inflammatory lesion about the size of a pea. There was an underlying infarct which affected the outer two-thirds of the ventricular wall, the surface being thickly coated with a fibrinous exudate. The artery had ruptured, and the pericardial sac was distended with blood. In our series of 57 cases of acute endocarditis, pericarditis occurred in 15. In pneumonia, pericarditis

¹ POST-MORTEM STATISTICS.

	Nephritis.				Amyloid Kidney.		Cirrhotic Kidney.	
	Cases.		Pericarditis.		Cases.	Peri-carditis.	Cases.	Peri-carditis.
Dickinson ²	39		2.5%		48	6.2%	68	23.5%
Grainger-Stewart ³	28		7.0%		50	8.0%	13	7.5%
	Acute Nephritis.		Chronic Nephritis.					
	Cases.	Peri-carditis.	Cases.	Peri-carditis.				
Sibson ⁴ ..	21	14.2%	62	9.6%	22	9.0%	162	14.1%

² Dickinson, W. H., *Diseases of the Kidney*, Lond., 1877, ii., 277, 421, 499.

³ Stewart, T. Grainger, *A Practical Treatise on Bright's Diseases of the Kidneys*, Edin., 1871, Second Edit., 171.

⁴ Reynolds's *System of Medicine*, Lond., 1877, iv., 407.

is usually trivial and terminal, an indication of a general septicæmia ; but it may occur early, and be severe. In only one of our cases was it gross, the exudate being mainly fibrinous and due to the pneumococcus, though gonococci were present in an urethral discharge, and the diphtheria bacillus in the throat ! Of the cases associated with renal disease, one was due to a tubercular infection, while one of those accompanying tuberculosis was due to a streptococcus.

TABLE SHOWING THE ASSOCIATION OF ACUTE PERICARDITIS WITH OTHER DISEASES.

	Hirschfelder ¹ (Johns Hopkins Hospital).	Personal.
Endocarditis	53	28
Myocarditis	8	—
Pneumonia	39	8
Acute rheumatism	31	—
Nephritis	33	14 ²
Tuberculosis	25	5 ³
Pleurisy	17	1
Gonorrhœa	3	—
Aneurysm	2	—
Leukæmia	2	—
Syphilis	1	—
Sepsis	—	3
Totals	214	59 ⁴

In the exceptional case the pericarditis is apparently primary, as no other lesion can be discovered. This was so in the case of a labourer who was at work in the morning and ate his midday meal with his usual appetite. He had no work in the afternoon, and went for a walk with some friends and drank a fair amount of alcohol. At half-past four o'clock he suddenly experienced acute pain in the front of his chest. The pain persisted, and friction sounds were detected a few hours later and remained audible for ten days. No other abnormality could be detected at the time, but on his dismissal a systolic murmur was audible at the apex. In another case with an equally sudden onset in a man who was in apparently good health, the pericarditis was found, on *post-mortem* examination, to

¹ *Diseases of the Heart and Aorta*, Philadelphia, 1910.

² One was tubercular.

³ One was pyogenic.

⁴ In seventeen of these cases the patients had suffered from acute rheumatism.

be due to a tubercular infection from a tubercular gland in the mediastinum.

The Symptoms of Pericarditis.—The symptoms of acute pericarditis vary considerably in different cases, as they may arise in various ways. In the early stages and in small effusions the interference with the cardiac action is slight, but in large effusions the mechanical interference is considerable. The amount of fluid may be large (as much as a gallon),¹ and much greater than a normal pericardium can hold (less than $1\frac{1}{2}$ pints); and although an inflamed pericardium stretches under the strain, the pressure within the sac always rises. Starling² has observed the results in experiment. On introducing oil into a dog's pericardium, the venous pressure steadily rose, though the aortic pressure at first remained constant; but a critical point was soon reached when the intra-pericardial pressure rose to such a height that it prevented the diastolic filling of the heart, and the supply of blood to the ventricles failed. The pulse then became small, and the blood-pressure rapidly fell.

The increasing difficulties can be readily observed as an effusion increases in size, the pulse rate becoming more frequent, and the volume and pressure lessening; while the surface becomes cyanosed and the respiration difficult. Hepatic engorgement is usual, but œdema is uncommon and is rarely extensive.

Pericardial inflammation has little direct effect upon the cardiac muscle, and lesions which are found *post-mortem* in uncomplicated cases are always trivial and confined to the superficial muscular layers. But pericarditis is rarely, if ever, uncomplicated. Endocardial lesions are often present, and a myocarditis not infrequently co-exists. Pleural or pulmonary disease may originate or accompany the pericardial inflammation.

The pericardium, too, is situated within the thorax, whose walls are capable of only limited displacement, and an increase in its size can only be accommodated by pressure upon the

¹ Gibson, G. A., *Diseases of the Heart and Aorta*, Edin. and Lond., 1898, 323.

² Starling, E. H., *Lancet*, 1897, i., 653.

neighbouring viscera. The lungs are compressed and emptied of their air, the œsophagus is driven against the vertebræ, the superior vena cava is obstructed, and the vagus, phrenic or recurrent laryngeal nerves may be implicated; and the symptoms in different cases vary accordingly.

The *onset* of pericarditis is more frequently attended by the occurrence of definite symptoms than is the case in endocarditis, and they generally suggest a cardiac origin; but pericarditis may be latent and only recognized on routine examination or *post-mortem*. This is most likely to happen when the patient is already seriously ill and insensitive to trivial discomforts, or in cases where a pulmonary affection interferes with auscultation; but even in sub-acute rheumatism pericarditis may be latent. A boy, aged eighteen, who was resident in hospital on account of an attack of acute rheumatism, suffered from a relapse of the arthritis of short duration and slight severity. Typical friction sounds and enlargement of the area of cardiac dullness were discovered on routine examination of the chest, though the pulse rate remained unaltered, and no thoracic discomfort was experienced.

In about half the cases (15)¹ the patient complains of *discomfort* or *pain* over the front of the chest. The pain is rarely severe, and may be general, or referred to the base of the heart or to the xiphoid cartilage, and varies in intensity at different times without any obvious cause. It is less frequent and less severe in dry pericarditis than in cases with effusion; and if present in the early stages, may become more marked or may remit with the occurrence of effusion. If the effusion is considerable, some complaint is almost invariable, and occasionally the anguish is extreme. The pain is generally increased by pressure with the stethoscope, but this probably depends upon the mobility of the chest wall, and is more constant in children than in elderly persons with calcified costal cartilages. In some cases the pain on pressure is due to tenderness of the skin, and even the slightest pressure with the stethoscope is resented, and poultices and fomentations cannot be borne; but this is exceptional. Palpitation is not uncommon.

¹ The figures in brackets show the incidence of symptoms in a series of 30 cases.

The *pulse* is almost always affected. In the early stages it is more frequent than normal, and out of proportion to the degree of fever. With the onset of effusion the rate increases and the volume and pressure diminish in corresponding degree to the pressure exercised upon the heart. The *pulsus paradoxus* may be present, but seldom in characteristic degree. The rhythm of the heart has never, in our experience, altered, though irregularities have often been described. Pericarditis is generally met with in cases where other cardiac lesions co-exist, and frequently occurs in persons whose pulse is already irregular, so that extrasystoles, auricular fibrillation, etc., are not rare; but these are seldom, if ever, the result of the pericardial inflammation.

Shortness of breath is quite as frequently present (18) as *præcordial pain*. It may of course be due to associated pulmonary or pleural disease, but it occurs also in uncomplicated cases. It is never extreme in cases without effusion, and is usually severe in corresponding degree to the amount of fluid, and, if this is considerable, may be accompanied by *orthopnea* (11). Sometimes the breathlessness is paroxysmal and occurs mainly at night, or is of the Cheyne-Stokes type; but this is probably always due to accompanying renal disease, which was present in the 3 cases of this series in which it was observed.

The other symptoms which may be present are all less constant. The facial aspect may be changed, the countenance becoming anxious and unduly pale (2), or distinctly cyanosed (9). This is most usual in effusion, and the degree of cyanosis in particular may be taken as an index of the degree with which the effusion is interfering with the cardiac work.

Difficulty in swallowing (1) or choking attacks (1) may be present in cases where the effusion is large and presses upon the *œsophagus*. The difficulty is usually increased if the patient is recumbent, and is relieved by the erect position. The voice may become weak and lost if the laryngeal nerves are specially implicated. The veins of the neck may be turgid and distended, and the face and head may be swollen and puffy. The other systemic veins, too, may be congested; but *œdema* (1) is rarely extensive. Hepatic enlargement may

be distinct, and accompanied by tenderness on pressure in the epigastrium.

In serious cases the patients are drowsy, or restless and sleepless, and are often mildly delirious at night. Acute cerebral symptoms occasionally ensue. Fever is inconstant. In acute rheumatism the temperature rises on the onset of pericarditis, but the rise may be inconsiderable and hyperpyrexia is rare. In renal cases and in terminal pericarditis fever is generally slight or absent.

The *course of events* in an attack of pericarditis varies greatly in individual cases, for the causal factors differ widely, and it may be a minor accompaniment of a serious ailment in which other viscera are gravely involved, or the chief episode of a purely cardiac illness. In pneumonia, for example, pericarditis is generally merely an indication of a widespread septicæmia and an additional cause of cardiac strain; in pyæmia it is but an item in a widespread sepsis; and in renal disease an indication of the way in which death is approaching, rather than its cause. In all three cardiac weakness is often present, and the advent of the serous inflammation merely accentuates the pre-existing symptoms, and it is apt to be missed unless routine examinations are frequently made. Even in the cardiac group associated with acute rheumatism its effects are often indistinct, for endocarditis and myocarditis may co-exist, and the exact influence of each factor can rarely be measured. The amount of the effusion cannot be accurately gauged by the size of the area of cardiac dullness, for this may be enlarged from hypertrophy consequent upon a valvular flaw, from dilatation on account of loss of tone, or from the presence of effusion, or may be the sum-total of all three factors. In the majority of cases the symptoms are only in part due to the pericarditis, their character is necessarily inconstant, and their progress varies within wide limits.

The anatomical distinctions between dry pericarditis, effusion, and adhesion are also only accurate in a general way, and every case does not run the full course. Friction may be audible for days without the occurrence of any appreciable effusion. Thus, in a man, aged seventy-two, who died in the wards from bronchitis and renal cirrhosis, it was constantly present from February 22nd until April 9th, while the peri-

cardial sac on *post-mortem* examination only contained four ounces of fluid. Fluid may be poured out rapidly, and within three or four days exert an obvious mechanical influence upon the cardiac action, or may appear and disappear insidiously and gradually, results which must be due to variations in the virulence of the bacterium and in the resistance of the individual. The effusion in rheumatic cases is usually sero-fibrinous and rarely purulent; in tubercular cases, in cancer, and in scurvy, it is often hæmorrhagic; and pus is likely to occur in streptococcic infections. And while a purulent effusion generally terminates fatally, as in the comparable empyema, unless operative interference is successfully undertaken, the recoveries which occasionally ensue in the latter disease suggest that a similar result may obtain in purulent pericarditis, a suggestion which is borne out by the discovery in a few cases of cretaceous material within the sac.

The relative frequency of the various forms of pericarditis is indicated by the following table, which shows their incidence in the *post-mortem* room:

Sero-fibrinous	..	..	..	..	..	..	108 ¹
Hæmorrhagic	..	..	..	..	..	..	30
Purulent	..	..	..	..	..	..	24
Tubercular (secondary)	..	..	..	..	..	..	24
Tubercular (primary)	..	..	..	..	..	..	2
Partially adherent	..	..	..	..	..	..	111
Totally adherent	..	..	..	..	..	..	23
Ossified	..	..	..	..	..	..	2
							324

In acute rheumatism the onset of pericarditis is most usually rapidly followed by effusion, which, within a few days (as a rule three or four, rarely a week) attains a maximum, and then gradually disappears. The process can be followed more or less accurately from day to day by percussion, the area of dullness steadily increasing and then lessening. Considerable differences, however, occur in the duration of the effusion. In a few cases the area of cardiac dullness regains its normal limits within a fortnight, but in others not for many weeks. An exudate, which is mainly serous, is rapidly removed, but a thick fibrinous deposit necessitates the formation of granula-

¹ Breitung, quoted by G. A. Gibson, *Diseases of the Heart and Aorta*, Edin. and Lond., 1898, 314.

tion tissue, and months may elapse before any appreciable alteration in the area of cardiac dullness can be detected. In a large majority of cases recovery ensues spontaneously, unless the endocardial lesions are widespread and actively progressive.

Sir William Osler stated that dry pericarditis does not kill, though it frequently occurs in dying patients, and the dictum is probably accurate. In effusion, too, the actual mortality is probably small, and the gravity of the individual prognosis depends chiefly upon the amount of the effusion and the degree of pressure exercised upon the heart ; but we have seen only three or four cases in which death seemed to result in this way. The mortality of pericarditis, however, is great (39·8 per cent., G. A. Gibson), so that its advent is always a herald of evil omen, even though it only acts indirectly.

In exceptional cases pericarditis is chronic, or, at any rate, sub-acute. The duration of the illness is prolonged, and on *post-mortem* examination, a soft, succulent exudate is found loosely uniting the two layers. In tubercular cases, the progress may be sub-acute, and much caseous matter may be found, the parietal and visceral layers being separated at times by half an inch of caseous material.

The Physical Signs of Pericarditis.—In *fibrinous* pericarditis the characteristic friction may be felt by the hand, but it is usually apparent only on auscultation. The sounds are superficial, and seem to originate close to the ear. They are usually double (to and fro) and of cardiac rhythm, but they do not correspond closely to the cardiac movements or sounds, occurring a little after systole has commenced, and continuing till a short period before the cessation of diastole, with an interval between the systolic and the diastolic portion ; and covering rather than replacing the cardiac sounds. The sounds may be of varying quality, very soft or harsh and grating, or scratching, or creaking like the creaking of new leather. The systolic element is usually louder and longer than the diastolic. Sometimes the sound is single and systolic in rhythm, and it may be triple if the auricular contractions impart a special emphasis at the end of ventricular diastole. The friction is usually loudest between the apex and the xiphoid

cartilage, and may be audible all over the cardiac area and even a little beyond it, or may be limited to a small area. It is occasionally only audible towards the base, or more rarely at the apex. It is variable in its distribution and character, which may change from day to day. It is often intensified by pressure with the stethoscope over an interspace, and by alterations in the respiratory phases, but it may be loudest during either inspiration or expiration; it may be unaffected by respiration. The sounds may vary in character and site with changes in position. There are no lines of conduction outwith the cardiac area, as in aortic valvular disease, and the sound is rapidly lost beyond the cardiac borders.

There is as a rule little difficulty in recognizing the nature of the sounds. They may resemble the murmurs of double aortic disease, but these are *constant* in character and site, and are conducted in definite directions. Pleuro-pericardial friction rarely occasions difficulty. It occurs when the pleural spaces overlying the heart are involved, when the friction may be cardiac in rhythm; but full inspiration or full expiration almost always abolishes the sounds, while in pericarditis the friction never disappears, even though the respiratory movements alter its quality. It is only audible at the periphery of the cardiac area, especially about the upper and left borders, and it is generally accompanied by evident pleural friction elsewhere.

Difficulty may be experienced when the sound is single, but its quality may be characteristic, and its variations from day to day separate it clearly from an endocardial murmur.

In *large effusions* in thin persons the physical signs are distinct. The præcordia may be prominent if the chest wall is soft, and the interspaces may be flush with or even bulging above the ribs, and the overlying skin may be œdematous. The apex impulse becomes weaker, and may disappear, though pulsation may be apparent in the fourth interspace; and the expansion of the left chest on inspiration is less free than that of the right. The movement of the diaphragm, too, is less ample, in particular upon the left side, and the liver and spleen may be evidently displaced downwards. The area of cardiac dullness is increased in all directions. The upper border rises above the third rib, and may extend as high as

the first, the maximum elevation being over the sternum. The left border extends outwards, and may be an inch or more beyond the apex impulse, if that still persists, and the right border extends towards or as far out as the right nipple. The shape, too, is altered, and it is conical instead of rectangular, the maximum diameter being at the xiphoid level (Figs. 243, 244).

The fifth right interspace is encroached upon early, and the cardio-hepatic angle is obtuse instead of right-angled, as in enlargement of the right auricle. The friction sounds usually persist, but their area of distribution becomes circumscribed.

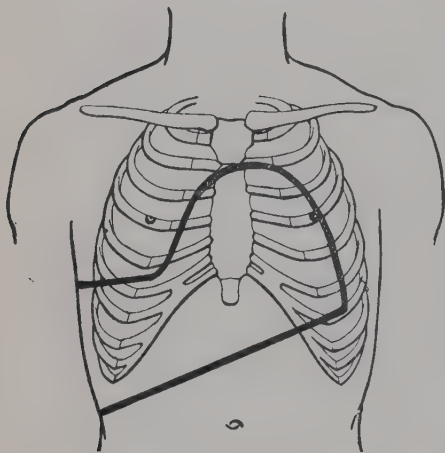


FIG. 243.—DIAGRAM SHOWING THE SHAPE OF THE AREA OF DULLNESS IN PERICARDIAL EFFUSION.

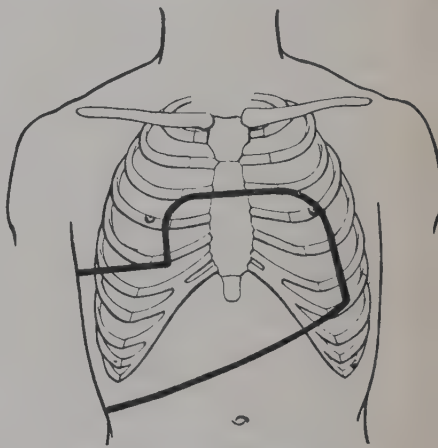


FIG. 244.—DIAGRAM SHOWING THE SHAPE OF THE AREA OF DULLNESS IN DILATATION OF THE HEART.

They are generally audible over the base of the heart, or rarely at the extreme apex; but they are sometimes little altered if the heart retains its relationship with the front of the chest. The cardiac sounds become weak, muffled and indistinct, and in large effusions may be short and very distant.

Some other signs have been described, but their occurrence is only occasional. The shoulder girdle may be raised, so that the finger can be introduced between the left clavicle and the first rib close to the sternum. An area of dullness, with diminished respiratory murmur, may be found at the extreme left base close to the vertebral column; and tubular

breathing, with ægophony, may be recognized at the angle of the left scapula.

In thin subjects the diagnosis of pericardial effusion is usually made without difficulty, but it may be difficult in very fat people, especially if the case only comes under observation at a late stage. In dilated hearts pulsation tends to become more widespread, though less ample and of shorter duration, rather than to disappear. The area of dullness is increased laterally rather than upwards, and in mitral stenosis alone reaches as high as the second rib, unless the heart is displaced upwards by abdominal distension; and the cardio-hepatic angle is a right angle. The sounds are generally short and sharp and superficial, and are often loud rather than distant and indistinct. The liver and spleen, though frequently enlarged, are not grossly displaced.

The physical signs may, however, mislead. As a rule, with the occurrence of effusion, the fluid collects between the heart and the front of the chest, so that the apex impulse disappears, the friction area diminishes, and the sounds become distant. But the heart may retain its position in front, while the fluid accumulates elsewhere. In one case under our observation a well-marked apex impulse persisted unchanged during the progress of an effusion, and the friction area remained constant, as the apex was firmly anchored to the adjacent pericardium by a thick, short band, the residue of an antecedent pericarditis. In another case the right border of the area of dullness remained immobile as the right pleural sac was obliterated and adherent to the chest wall. And in a third the area of dullness diminished in size and moved steadily to the right side from the gradual development of a left pneumothorax.

The pulse is not an infallible guide to the degree of the effusion. As a rule with an increasing effusion the pulse steadily becomes more frequent, quick and soft; but its character may be disguised by arterial thickening, aortic regurgitation, or antecedent high blood-pressure. The shape of the distended pericardium, as shown in a radiogram (Fig. 245), is characteristic; but examination of this kind is rarely possible.

The onset of pericarditis may be latent, and the diagnosis may be made on routine examination. In the majority of

cases, however, complaint of pain in the cardiac region, or an exacerbation of the fever, attract attention, and are associated with an increase in the rate of the pulse. The latter is rarely absent, and, as in acute endocarditis, the pulse rate is increased out of proportion to the fever and the number of respirations, the pulse-respiration ratio becoming 5, or even 6, to 1. With the occurrence of effusion, the pulse rate still

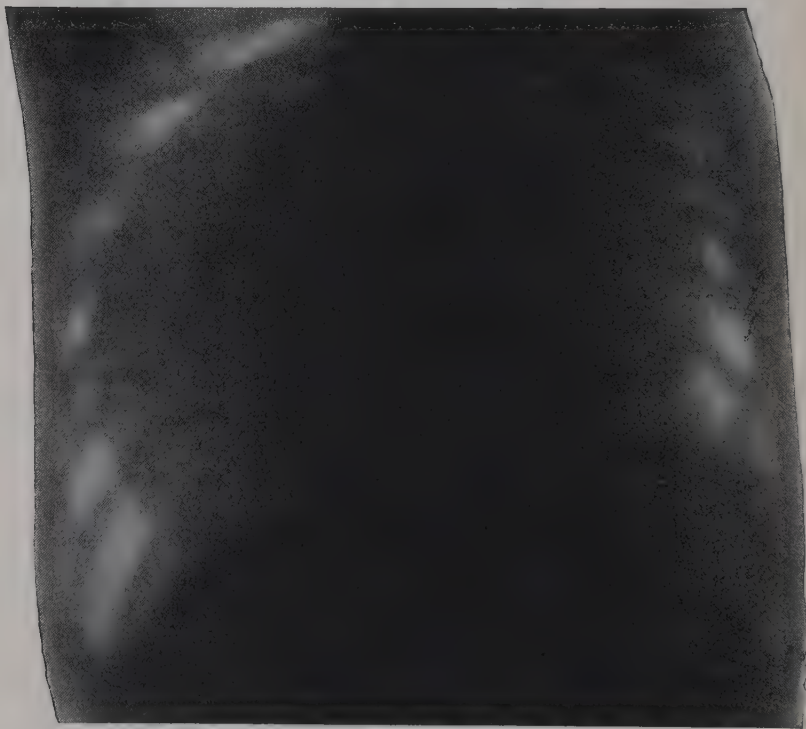


FIG. 245.—RADIOGRAM: PERICARDITIS WITH EFFUSION AND LEFT PNEUMOTHORAX (J. R. R.).

further increases, the respirations become more frequent, and the extraordinary muscles of respiration are brought into play, while the mucous membranes become cyanosed, and the patient is forced to sit up in bed. With an increasing effusion, the distress is rapidly augmented, the countenance becomes anxious and distressed, the cyanosis deeper, and the pulse and respirations still more frequent. In the later stages the

cardiac exhaustion steadily increases, the pulse becomes smaller and more quick and of lessened pressure, the cyanosis is combined with an ashen pallor, and the patient tends to sink downwards in the bed, his skin often bedewed with a cold sweat; and death ensues from cardiac failure.

In the individual case, attention should be paid to the features which have just been mentioned. The occurrence of orthopnœa, unassociated with any pulmonary lesion, a definite diminution in the volume and strength of the pulse, and the combination of pallor and cyanosis, are all of evil omen. The degree of pyrexia is of little importance, but the rate of the pulse and of the respirations convey their special indication of cardiac or respiratory embarrassment. Failure to maintain the erect posture, if unaccompanied by definite evidence of improvement, is a most serious symptom.

CHAPTER XXV

ADHERENT PERICARDIUM

THE term "adherent pericardium" is generally used somewhat loosely, and comprises all cases in which local or universal adhesions exist between the visceral and parietal layers of the sac. *Mediastino-pericarditis* includes two groups: one in which there is a marked increase of the fibrous tissue in the mediastinum, with pericardial adhesions, and with adhesion of the exterior of the pericardium to surrounding parts (*indurative mediastino-pericarditis*); and a second in which the parietal pericardium is adherent to surrounding parts, with pericardial adhesions, but with little or no general mediastinitis (*pericarditis interna et externa*). *Chronic mediastinitis* implies that there are no internal pericardial adhesions.

Pericardial adhesions necessarily entail additional work for the heart, but this may be insufficient to produce appreciable hypertrophy if they are tenuous and lengthy and limited in their distribution. Sibson¹ collected 50 cases in which the size of the heart was normal. If, on the other hand, the adhesions are thick and extensive, and if, in addition, the inflammation has spread outside the sac and fixed the heart to adjacent parts and limited its free movement, hypertrophy is always considerable and of appreciable degree. When pericardial adhesions and valvular disease co-exist, the heart is always hypertrophied. Sibson¹ found that hearts with valvular defects and adhesions were definitely heavier than hearts in which comparable valvular defects were present without adhesions; so that the latter must be held responsible for some part, at any rate, of the hypertrophy.

Local adhesions are most frequently found at those parts of the heart which alter their relations to the parietal peri-

¹ Sibson, F., *Reynolds's System of Medicine*, Lond., 1877, iv., 440.

cardium in least degree during the cardiac movements. They are thus most common near the apex and about the left border of the left ventricle, over the left auricle, and at the basal regions of the great vessels; and are less common over the right ventricle, the right auricle, and the origins of the great vessels.

The extra-pericardial adhesions also vary in their site and extent, and the heart may be adherent to the lungs, the diaphragm, the sternum and adjacent ribs, or even to the vertebral column. The inflammatory spread is sometimes minimal and sometimes local, and the symptoms and signs correspond accordingly. Pericardial adhesions, too, may vary in their effects upon the different chambers of the heart. They may surround and entangle the auricles and great veins and hinder the influx of the blood; or embrace the ventricles and the arteries and impede its exit from the heart. If the pleural cavities are obliterated, or the heart is adherent to the parietes, the respiratory movements will affect the cardiac action.

The etiology of adherent pericardium is presumably similar to that of acute pericarditis, but the chronicity of the affection makes it difficult to appreciate the various factors at their proper value. In Sibson's series 49 out of 80 cases had valvular lesions, with as a rule some form of chronic renal disease. In 3 cases aneurysm of the aorta was present, and in 1 cancer of the heart. In 19 cases the valves and aorta were healthy, and in 7 of the 19 no other viscus was diseased. In 22 cases which we have observed 13 had valvular disease, of whom 4 had renal disease in addition; and in 9 the valves were normal, of whom 4 had renal lesions; 4 cases suffered from tuberculosis; 1 from a chronic pulmonary fibrosis, which probably originated in an acute pleuro-pneumonia accompanied by empyema; in 1 the origin was unknown, as it was discovered in a patient who died from acute pneumonia; and in 1 it arose during acute rheumatism, though no valvular lesion was found *post-mortem*.

In Harris's series of cases of mediastino-pericarditis examined *post-mortem*, 11 seemed to be due to tuberculosis, 2 to acute rheumatism and enteric fever, and 1 to chronic bronchitis, syphilis, scarlatina, and trauma. In 6 the origin was not

apparent, and the symptoms commenced insidiously without any obvious acute thoracic disease.

The symptoms in adherent pericardium depend upon the degree to which the adhesions interfere with the cardiac action. In many cases symptoms are absent, as from their extent or from their character the adhesions do not incommode the heart. In other cases the interference is considerable, but the co-existence of valvular disease in sufficient degree to account for the symptoms which are present makes the recognition of the pericardial element difficult if not impossible. Thus in the majority of cases the condition is first recognized *post-mortem*, unless the previous existence of an acute pericarditis is already known.

In cases, however, where adhesions exist outside the pericardial sac, as well as within it (mediastino-pericarditis) symptoms of cardiac insufficiency are generally manifest, as they entail a very considerable amount of extra work on the heart, and so curtail the reserve to an appreciable degree. Sequeira has suggested that the ill effects may originate in another way from that which has been already discussed. The pericardium is strong and indistensible, and under normal conditions prevents undue distension of the cardiac cavities under the stress of physical exertion. In acute pericarditis its resistance may be seriously impaired by inflammation, and its cavity may be grossly distended by effusion, and its functions may thus be seriously compromised; a result which is more likely to follow if physical exertion has been undertaken before the inflammation has completely resolved. Dilatation of the heart may thus arise and persist; and if the sac, when distended, contracts adhesions to the neighbouring viscera, and is unable to return to its former dimensions when the inflammation has subsided, the results will be still more serious.

In this latter group of cases with inflammatory adhesions outside and within the pericardial sac, a characteristic syndrome (*Pick's* or *Concato's disease*, polyorrhomenitis, polyserositis) is not uncommon. The majority of these patients suffer from ascites and enlargement of the liver with, in addition, evidences of cardiac failure and sometimes effusions into the pleural

cavities as well ; jaundice may occur, but is rare. The abdominal symptoms are probably due to chronic peritonitis, which is most marked upon the diaphragmatic surface of the liver, accompanied by a varying degree of hepatic cirrhosis ; but the latter may be, as in one of Harris's cases, wholly absent even when ascites is a prominent symptom. In some cases the earliest symptoms are cardiac (as in Case 1, *see* p. 435), and the inflammation originates within the pericardium. The sequence of events in the early stage may be apparent on *post-mortem* examination. A man, who was dying from a chronic nephritis, developed an acute pericarditis ten days before his death. There was a well-marked fibrinous pericarditis, and the inflammation had spread through the diaphragm to the left lobe of the liver, which was attached to the central parts of the diaphragm by soft fibrinous adhesions. In others the primary lesion may be pleural, or both the pleural and pericardial lesions may occur simultaneously. The combination is by no means unusual, and occurred in 3 of our cases of acute pericarditis ; while in 6 others pleurisy developed either before or after the pericarditis.

In our series of 22 cases, a diagnosis of pericardial adhesion was made in 6, 3 of which are reported below. In two the diagnosis was based upon the previous observation of the acute stage. In the sixth, a patient who died from cardiac failure, the evidences of congestion in the area of the superior vena cava were persistent and in excess of those in the area of the inferior cava. The pericardial and the right pleural sacs were obliterated by adhesions, which in some parts were very thick, the mediastinum showed evidences of chronic inflammation, and the superior cava was narrowed to nearly half its diameter immediately above the right auricle.

The Physical Signs.—In many cases no evidence of pericardial adhesions is apparent, and the diagnosis is made on *post-mortem* examination. This is most frequently the case when valvular disease co-exists, as the symptoms are naturally ascribed to the valvular defect. A knowledge of the previous occurrence of acute pericarditis may suggest the existence of adhesions, but it must be remembered that the signs of “ adherent pericardium ” are really those of chronic mediastino-

pericarditis, and do *not* follow mere adhesion of the visceral and parietal layers.

In a general way the *apex impulse* is diffuse and forcible and displaced; pulsation is also evident in the epigastrium, and to the left and often to the right of the sternum; the latter as well as the ribs may pulsate visibly. In children the præcordia may be prominent. The apex impulse may be fixed to the chest wall and show little or no alteration in site, when the patient turns on to his left side; or in force, during full inspiration, if the pleural cavity is also obliterated. But these signs are really evidence of pleural, and not of cardiac, adhesion, and have been found to be present in cases where the pericardium was quite normal. Immobility of the lateral borders of cardiac dullness, especially on the left side, during the respiratory movements, owns a similar origin, and though generally associated with pericardial adhesions, may lead to a false conclusion.

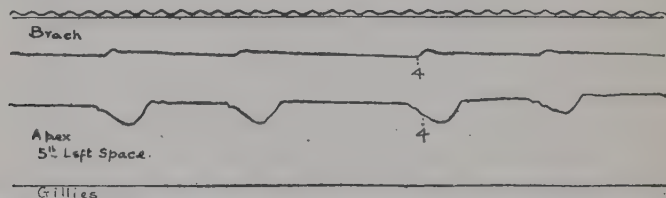


FIG. 246.—CURVES FROM THE BRACHIAL ARTERY AND APEX IMPULSE, SHOWING SYSTOLIC RETRACTION OF THE IMPULSE.

The normal systolic protrusion of the apex impulse may be replaced by systolic recession (Fig. 246). The explanation is somewhat difficult to understand, for the true (left ventricle) apex impulse must protrude during systole, as the longitudinal axis of the heart is always lengthened. It seems probable that systolic retraction of an interspace in the vicinity of the apex impulse is due to enlargement of the right ventricle pushing the left ventricle away from the front of the chest. Pulsation which is due to the right ventricle is negative (*see* p. 309), and in these cases becomes perceptible on account of the adhesions to the chest wall. Similar recession of the interspaces close to the sternum is often seen in persons with hypertrophied hearts, as the transverse diameter towards the base of the ventricle is always shortened during systole, and

the atmospheric pressure upon the surface of the chest causes a recession. The sternum too may be retracted during systole (Fig. 250); this is probably always a sign of adhesion.

A similar systolic recession may be found to the right of the sternum in the fourth and fifth interspaces from similar causes, and even the cartilages may be retracted if they are soft. Here, too, a fallacy obtains. Fig. 247 was taken from a patient with mitral stenosis, in whom *post-mortem* examination showed

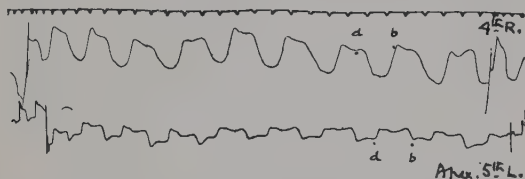


FIG. 247.—CURVES FROM THE FOURTH RIGHT INTERSPACE AND APEX IMPULSE, SHOWING SYSTOLIC RETRACTION OF THE FORMER.

neither pericarditis nor mediastinitis, so that the sign is obviously inaccurate. Broadbent has described “visible pulsation, synchronous with the cardiac systole, of the left back in the region of the eleventh and twelfth ribs, and in less degree of the same region on the right side,” as strongly suggestive of the adhesion of the heart to the diaphragm, the cardiac contractions pulling the diaphragm upwards and so drawing

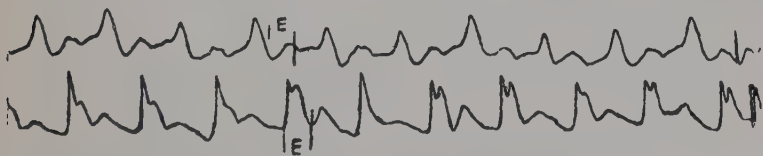


FIG. 248.—CURVES FROM THE LEFT BACK IMMEDIATELY BELOW THE COSTAL MARGIN, AND JUST OUTSIDE THE LINE OF THE SCAPULAR ANGLE, AND THE RADIAL ARTERY, SHOWING THAT THE RETRACTION IN THE FORMER SITUATION IS SYSTOLIC IN RHYTHM (BROADBENT'S SIGN).

the false ribs inwards with each systole; a similar pulsation may be apparent on the front of the chest at the anterior ends of the sixth and seventh ribs. But he has subsequently found the sign to be present in cases of hypertrophied heart without adhesion. The pericardium was normal in one case in which we have observed it (Fig. 248).

Adhesion of the heart to the diaphragm or to the sternum

may prevent or limit their movements during respiration. The defect in abdominal movement is readily appreciated by the eye or by the hand. Deficiency in the respiratory movement of the sternum is best shown with the calipers, for the increase in the antero-posterior diameter of the chest is slight, rarely exceeding 1 inch in normal individuals, and is always limited in emphysema and in pigeon-breast.

The pulse is generally rather small and somewhat frequent in cases which come under observation on account of cardiac insufficiency. It frequently varies both in volume and force with the respiratory movements, becoming larger and stronger during expiration (pulsus paradoxus, Fig. 249), the exact oppo-

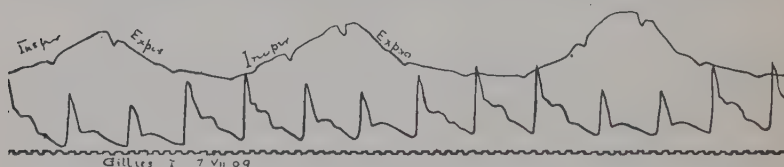


FIG. 249.—CURVES OF RESPIRATORY MOVEMENTS AND RADIAL PULSE : PULSUS PARADOXUS.

The pulse is largest during expiration.

site of the normal, but this has been observed in pericardial effusion, and even in cases where the heart was normal. Failure of the pulse during full inspiration, however, does not seem to occur unless mediastino-pericarditis is present. Pulsus paradoxus is probably due in these cases to adhesions about the root of the heart, embracing the aorta and the great veins ; and to traction exerted upon the fibrous bands during inspiration narrowing their lumen and so limiting both the output and the intake of the heart. Inspiratory filling of the cervical veins (Kussmaul's sign) owns a like cause, and may also be present.

The pulse may, of course, present the characteristic features of that of aortic incompetence or renal cirrhosis, should either of these conditions co-exist.

The other signs which have been described are less frequently present, and may be due to many causes. Sir W. Broadbent drew attention to the fact that the diastolic shock of the arterial valvular closure was often well marked and felt over a large area ; but this may also obtain in dilatation of the aorta and in

high blood-pressure. Riess has described cases in which the cardiac sounds were of metallic quality and well conducted to the skin surface overlying the stomach. Sewill has reported reduplication of the first sound ; and Thayer thinks that the third cardiac sound is usually very distinct. All these signs, however, are infrequent in adherent pericardium, and may be present in other cardiac abnormalities.

None of the signs ascribed to pericardial adhesions are thus pathognomonic, and they may all be absent in individual cases.

A patient died recently in the wards from valvular disease of the heart who had previously suffered from an attack of acute pericarditis of rheumatic origin under our observation. A week before her death we carefully examined her for all the signs of adherent pericardium which have been described above, with the exception of Reiss's sign and immobility of the apex impulse on change of position. All were absent, but *post-mortem* examination showed that the pericardial sac was entirely obliterated by fairly firm adhesions. The heart, however, was not adherent to the chest wall, mediastinitis was minimal and in no way indurative, and the pleural cavities were normal.

The previous existence of pericarditis ; the presence of several signs in conjunction, in particular a well-marked systolic retraction of a powerful apex impulse, or of the sternum ; immobility of the impulse and of the borders of cardiac dullness during respiration and during change of position from left to right side ; and the occurrence of cardiac insufficiency without any other obvious cause, are strongly in favour of the presence of mediastino-pericarditis. Examination with the Röntgen rays is often valuable. The mediastinal induration may be apparent in radiograms (Fig. 251), and limitation of the movement of the diaphragm from the presence of adhesions may be visible with the screen.

The following cases illustrate the usual symptoms of Pick's disease :

CASE 1.—A boy, aged sixteen, was admitted into hospital on May 11th, 1908, complaining of breathlessness and of pain in the left side of the chest, and of swelling of the abdomen and of the face and feet.

He had always been healthy, save for an attack of scarlatina in childhood, until he contracted rheumatic fever four months before

admission. Many joints were affected, and he sweated profusely, and was confined to bed for three weeks. Even when he was lying in bed he was very breathless, and this continued for a month after he got up, being intensified on exertion, and he spent the greater part of this time in bed. The dyspnoea then lessened, but he was still unfit to return to his work at the pit. Three weeks before admission, the lower eyelids and the feet became swollen, and the abdomen was noticed to be enlarging, and he was again forced to return to bed. He had a slight cough without any expectoration, and his left side became painful at times, but otherwise he had no special discomfort, and his appetite was good.

On admission, he was found to be a well-grown lad of fair nutrition. He lay flat in bed, by preference on his right side.

There was slight œdema in the legs and over the sacrum, but none in the face, and his complexion was somewhat pale. The tongue was coated and rather dry, and the pharynx was congested. The abdomen was considerably distended and contained a large quantity of free fluid. The spleen could not be felt, but the liver was definitely enlarged. Rhonchi were audible all over the chest and râles at the posterior bases.

His progress thereafter was satisfactory. The œdema and ascites rapidly disappeared, and he left hospital on September 12th, 1908, in fair health, and resumed his work as a collier. He continued at work until March, 1909, when the abdomen again began to swell, and as he became unable to stoop he had to cease work. There was no recurrence of the general œdema, and no other symptoms were present beyond slight breathlessness on exertion; but the ascites increased, and he was again forced to go to bed in July.

On admission in August, he seemed to have lost flesh, and he was pale and thin. He lay easily in bed and made no complaint, though the ascites was now much greater than on his first admission, but there was no œdema of the feet. His progress, however, was not satisfactory. The ascitic accumulation had to be removed frequently, as it rapidly recurred after tapping. The fluid presented the characters of a transudate, the cellular elements being mainly lymphocytes, with some large endothelial cells, which were often *en plaque*, and a few polymorphonucleated cells. In December he was transferred to a surgical ward for operation. Death ensued suddenly, when he was under the anæsthetic, before any operative interference had been undertaken.

The physical signs which were observed during life were not very distinctive evidence of adherent pericarditis. There was no pulsation on the front of the chest, though the apex impulse could be felt indistinctly in the fifth left space when he lay on his left side. The area of cardiac dullness was of normal site and size, and was not altered by changes in position or in respiration. The cardiac sounds were pure but short. On his first admission, murmurs were present from time to time, both at the apex and at the base, but they never persisted for any length of time; on his second admission the sounds were pure. The pulse was always quite regular, but a little frequent (90 to 100). The chest expansion measured 2 inches, and the diaphragm moved fairly

well with respiration. There was no inspiratory distension of the cervical veins, though there was a slight pulsus paradoxus, and there was no systolic drag upon the false ribs; but the liver was large, and steadily increased in size (the vertical measurement being $7\frac{1}{2}$ inches on his second admission), and the ascitic transudation with minimal cedema pointed to a hepatic origin. The antecedent rheumatism accompanied by breathlessness while at rest suggested, in the absence of gross pulmonary lesions, a cardiac origin, and a diagnosis of adherent pericardium and perihepatitis was accordingly made.

On *post-mortem* examination, the pleural cavities were found to be completely abolished by firm adhesions everywhere, save at the base of the right lung, where 2 pints of fluid were present between the diaphragm and the base of the lung. The pericardial surfaces were united by firm adhesions, which were fully half an inch thick in places, and the diaphragm was firmly adherent to both liver and spleen, the hepatic peritoneum elsewhere being only slightly thickened. The liver was considerably enlarged and showed an early, fine, monolobular cirrhosis. The cardiac valves were normal.

CASE 2.—A pithead worker, aged seventeen, was admitted to hospital on June 10th, 1911, complaining of swelling of the feet and legs of five months' duration.

His previous health had not been good. He had had whooping-cough and measles in childhood, and had fallen and broken two of the right ribs at the age of thirteen, and shortly afterwards was admitted into hospital on account of ascites with occasional attacks of pain in the right iliac fossa. In July, 1907, the abdomen was opened, and the intestines were found to be covered with miliary tubercles. He made a good recovery, and worked for eight months at the pithead, but the ascites recurred, and in July, 1908, as the scar of the former wound was thin and distended, the abdomen was again opened. The liver was now enlarged and rough, so an attempt was made to procure adhesions between it and the parietal peritoneum. On dismissal in September, his general condition had improved, but he was distinctly cyanosed and had a troublesome cough. He was unable to return to work as the cough continued, accompanied by a yellow sputum, and he was breathless, and felt his heart beating on any exertion, and cedema appeared at times in the feet and legs. In January, 1911, the cedema became permanent and gradually increased in extent, and the breathlessness became more marked.

On admission, the patient lay comfortably flat in bed. He was only moderately developed, and was poorly nourished, and there was widespread cedema, though it was not extreme. The mucous membranes were very blue, and the face was puffy, and the hands and feet were congested and purple. The malar capillaries were distended and prominent. The tongue was moist and coated. The abdomen was large but not tender. The parietes were cedematous, and there was slight ascites. The liver was enlarged, but not tender, the vertical measurement being $6\frac{1}{2}$ inches, the edge and surface being smooth and firm;

it moved very slightly with respiration, the movement on the left side being more ample, though distinctly less than normal. The spleen was slightly enlarged. The respiratory murmur over the right lower back and axilla was deficient and accompanied by fine, bubbling râles.

The patient remained in hospital until November, but received slight benefit from his treatment. He made little complaint, and had few discomforts when he was confined to bed, but the œdema increased whenever he was out of bed for any length of time.

There was well-marked pulsation over the front of the chest, the whole præcordia heaving, though it was not unduly prominent. The apex impulse was fixed and was unaltered by changes in position or by the respiratory movements, and was *negative*, a systolic retraction; and the third, fourth, fifth, sixth, and seventh cartilages on both sides, but less strongly on the right, and the lower sternum (Fig. 250) showed similar movements, the interspaces moving more freely than the cartilages. The sternal pulsation was exaggerated on deep inspiration. The area of cardiac dullness was enlarged on both sides, and was not influenced by the respiratory movements. The cardiac sounds were pure, the first sound being everywhere short and sharp and loud. The pulse was always regular, but a little frequent (90 to 100). The blood-pressure was normal. There was distinct pulsus paradoxus, and the sternal movement forwards on inspiration was nil. Broadbent's sign was absent, and there was no visible distension of the cervical veins at any time.

A radiogram showed considerable mottling on each side of the heart, especially to the right. The right heart was enlarged, the cardio-hepatic angle was obtuse, and the upper margin of the diaphragm was indistinct.

The history of this case presents an absence of the usual causes of mediastino-pericarditis, and the injury to the ribs which preceded the onset of the ascites was the only illness known which might affect the thoracic viscera. The presence of diffuse tuberculosis in the abdomen at the time of the first operation, however, suggests another cause, chronic tuberculosis in the mediastinum; and the localization of râles to the right lower lobe alone and the diminution of the respiratory murmur pointed to a local pulmonary lesion, although no definite signs of a massive consolidation were present. No tubercle bacilli could be found in the sputum. There is in the Museum of the Western Infirmary of Glasgow a specimen of a heart with an enormously thickened (tubercular) pericarditis, which arose from the direct infection of the pericardium by a tubercular gland in the mediastinum. The whole of the mediastinum was matted together around caseous glands,

and a tuberculous pleurisy co-existed, so that the factors required for further spread were already present. In Harris's series of 25 cases, 11 seemed to be due to tuberculosis.

CASE 3.—A cabinet-maker, aged twenty-seven, was admitted to hospital on July 1st, 1909, on account of a right hemiplegia which had occurred suddenly two days previously.

His health had been good until two years before admission, when he became subject to "indigestion," which was occasionally accompanied by vomiting. His face was at times swollen, and he was short of breath, and for the last six months had been troubled with headache and swelling of the abdomen. Two months before admission he had to cease work for a fortnight on account of abdominal pain and swelling accompanied by headache and vomiting.

He was an unhealthy-looking man, of poor physique. The face was puffy and covered with acne, and the venules on the nose were dilated. The eyeballs were rather prominent, and there were slight nystagmoid movements when the eyes were turned to the side. The veins on the forehead were dilated and prominent, and the left jugular veins were large and full. The muscles were small and soft, and enlarged glands were present in the axillæ and groins. The tongue was coated, many of the teeth were carious, and the tonsils were enlarged and inflamed.

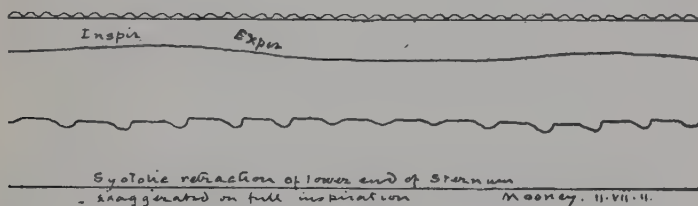


FIG. 250.—CURVES OF RESPIRATORY MOVEMENTS AND RETRACTION OF LOWER END OF STERNUM.

The abdomen was large, mainly on account of enlargement of the liver, which occupied the greater part of the right hypochondrium; the area of dullness measured $7\frac{1}{2}$ inches vertically. The edge was thickened and hard, and not tender, and moved but little with respiration. A small amount of free fluid was present in the abdomen. Improvement was rapid, and power steadily returned to the paralyzed limbs, while his speech regained clearness and accuracy.

In the middle of July he suddenly experienced acute pain in the right hypochondrium, and the pulse became more frequent. The liver was distinctly larger than it had been, and was now tender to palpation, and friction was audible over the lower part of the right chest. The symptoms subsided within ten days, the hepatic tenderness disappeared and the liver returned to its usual dimensions. Improvement continued, and he was able to go home on September 13th, walking

fairly well. His colour, however, had deepened, and he was more cyanotic than on admission.

He was readmitted to hospital on March 31st, 1910, on account of pain and swelling in the abdomen and cedema in the legs. The pain had recurred on five occasions in the interval. It was never very severe, but, commencing gradually, steadily increased for a week or so, and then gradually disappeared after ten days or a fortnight. The bowels were costive during the attacks, but there was no vomiting. The symptoms had been more severe since January, and the legs had become swollen.

On admission, he was more cyanotic than before, and he required three pillows on his bed. There was extreme ascites, and cedema in the limb which had been paralyzed though there was none in the other, and he was short of breath and incapable of any exertion.

The præcordia generally was prominent, and there was well-marked pulsation in the second, third, fourth, fifth, and sixth left interspaces, the maximum being in the fifth and negative, a retraction during systole (Fig. 246); and with diastole a well-marked shock was felt on palpation all over the right chest. The impulse moved for nearly an inch when he turned on to his left side, and decreased in force during inspiration. The area of dullness was of normal site and size, but the right border was unaffected by respiration. There was pulsation in the epigastrium, which was systolic in rhythm. All the veins of the body were prominent and full, and the valves stood out distinctly, the veins on the sides of the chest and the left external jugular being most distinct; one on the left flank was varicose. On auscultation, the sounds were everywhere pure, but everywhere triple. The first sound was somewhat loud, but short and sharp, and the emphasis was on the middle sound. The pulse was small and poorly sustained, and about the normal rate. During his first residence it was quite regular, save for occasional extrasystoles, but during his second stay it was always irregular (auricular fibrillation). A radiogram showed considerable fibrosis of the right lung, which was most marked at the base, where it was intimately mingled with the shadow of the diaphragm and the right border of the heart (Fig. 251). The lower interspaces of the chest were retracted on inspiration. The false ribs showed no cardiac movement. Slight pulsus paradoxus was present.

The diagnosis in this case seemed clear, but the cause was not very apparent at first. On examination, however, a scar was found at the lower part of the right chest behind where an "abscess" had been opened in childhood, and there was dullness and deficient respiratory murmur in this situation, so that it seemed probable that an empyema had been present. The recurring attacks of perihepatitis, however, suggested the existence of an active focus of infection, but none could be discovered beyond the tonsillar inflammation and a slight catarrh in the lungs.

For the next three years he was able to lead a quiet life in fair com-

fort, but towards the end of 1913 œdema appeared in the feet and persisted. His breathlessness, too, increased in degree. In July of 1914 his symptoms somewhat rapidly increased in severity and he was forced to stay in bed. In August he was re-admitted into hospital

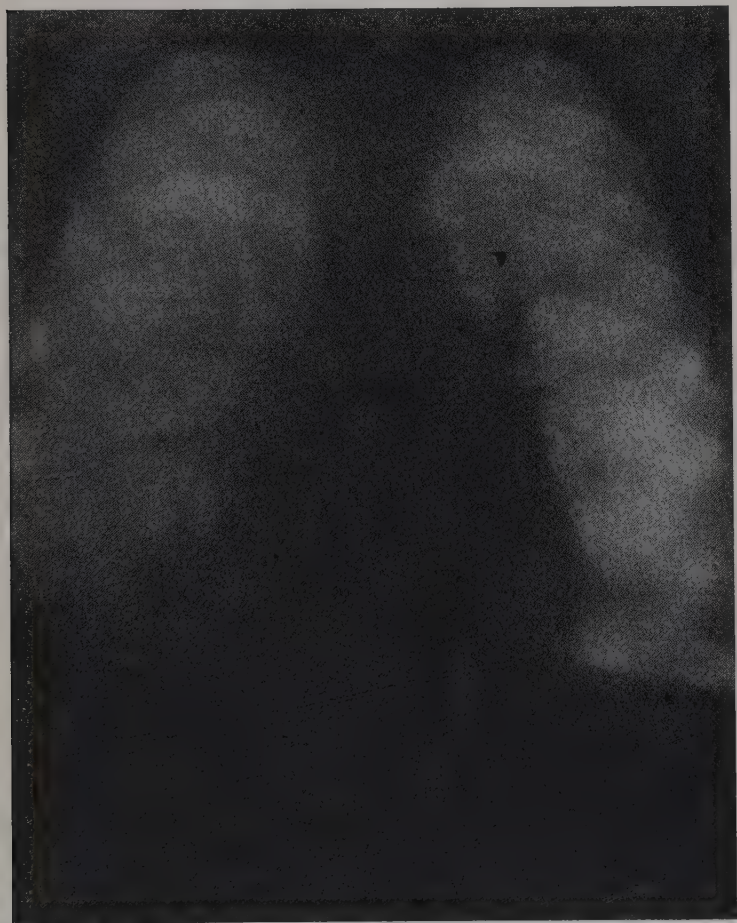


FIG. 251.—RADIOGRAM FROM A CASE OF MEDIASTINO-PERICARDITIS, ASSOCIATED WITH CHRONIC PLEURO-PNEUMONIA OF RIGHT LUNG (J. R. R.).

notably cyanosed, very breathless, and grossly swollen. He died somewhat suddenly next day. The *post-mortem* examination showed a chronic endocarditis of the mitral valve, and a greatly thickened pericardium, with extensive calcareous deposits in it, closely adherent to the whole surface of the heart and to the sternum, ribs, and pleura.

The right lung was extensively fibrosed, and the pleura thick and adherent. The left pleura showed a similar condition. The liver and spleen were imbedded in thick adhesions, and infarcts were present in the latter organ and in the kidneys.

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- SIR JOHN BROADBENT: *Adherent Pericardium*. Lond., 1895.
 THOMAS HARRIS: *Indurative Mediastino-Pericarditis*. Lond., 1895.
 PICK, F.: *Zeitschr. f. klin. Med.*, 1896, xxix., 385.
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Pneumo-pericardium.—The presence of gas within the pericardial sac is extremely rare. James,¹ when recording his own case in 1904, was able to find only 37 undoubted cases in the literature. To these we can add the three cases recorded by Ljungdahl,² Weil and Loiseleur,³ and Meyer,⁴ and that which is recorded below.

Pneumo-pericardium may conceivably arise in several ways. In a few cases it has apparently developed *post-mortem* from infection of the sac by a gas-producing organism; but no case has yet been recorded in which such an occurrence has been observed during life, and in only 5 of James's cases was the pericardium closed, though in Ljungdahl's and in our case no evidence of a breach of continuity was discovered.

In a second group the gas is due to the entrance of air through a wound. This has occurred in compound fractures of the thorax, stabs, gunshot wounds, and operative procedures such as paracentesis of the pericardial cavity, and has been produced as a therapeutic measure by Wenckebach⁵ in chronic pericardial effusion, apparently with benefit.

In a third group pneumo-pericardium is due to ulcerative processes connecting the sac with an air-containing viscus. The primary lesions have been varied; ulcerating cavities in the lung, both tubercular and gangrenous; pyo-pneumo-thorax; œsophageal ulceration, both cancerous and traumatic; and subdiaphragmatic abscess due to gastric ulcer, hepatic abscess, and even appendicitis.

¹ James, W. B., *American Medicine*, 1904, viii., 23.

² Ljungdahl, M., *Deutsch. Arch. f. klin. Med.*, 1913, cxi., 19.

³ Weil and Loiseleur, *Presse méd.*, 1916.

⁴ Meyer, A., *Med. Rec.*, 1918, xciii., 38.

⁵ Wenckebach, K. F., *Zeitschr. f. klin. Med.*, 1910, lxxi., 402.

In the majority of cases the lesion which permits the pneumo-pericardium produces in addition an infection of the sac, and effusion sooner or later develops, and very often becomes purulent. The symptoms and the physical signs are, in consequence, as a rule due in part to pericarditis, and in part to the aerial distension of the sac.

The onset of the *symptoms* may be gradual or sudden. In the former case the symptoms are those of acute pericarditis; in the latter the sudden occurrence of cyanosis, collapse, syncope, præcordial oppression or pain, denotes the advent of the new lesion.

The *physical signs* are usually striking. The apex impulse disappears, though it has been noticed to return if the patient assumed the prone position, and the area of cardiac dullness is replaced by an area with a tympanitic or skodaic note. In Stokes's case a cracked-pot note was present for a time. As effusion develops, the area of tympanitic percussion sound becomes diminished and varies in site according to the position of the patient. The *auscultatory phenomena* at this stage are characteristic, and the cardiac contractions produce the rhythmic churning, splashing, "mill-wheel" sounds, which are distinctive of the presence of air and fluid within the pericardium. In Stokes's case they had a peculiar metallic character; metallic tinkling has frequently been noticed. Friction sounds of the usual kind often co-exist.

In the majority of cases the diagnosis has been easy, as only 6 of James's cases were not recognized before autopsy. In our case, however, the condition was only diagnosed on examination with the X-rays, but in this instance the characteristic auscultatory and percussion phenomena were absent, as effusion did not develop, and the heart maintained throughout its normal relation to the front of the chest. The distension of the sac, too, at the first X-ray examination was slight. The diagnosis in Ljungdahl's case was also made with the X-rays, but no radiogram was taken.

The *prognosis* in pneumo-pericardium is better than might be anticipated, for 11 of James's cases recovered, so that 13 out of 40 cases did not die. In many of the fatal cases, too, the primary lesion was evidently likely to prove fatal.

The *treatment* of this disease must be conducted on ordinary

lines. Pyo-pneumo-pericardium suggests the advisability of surgical interference if the general condition of the patient permits.

We have only seen one case of pneumo-pericardium, and the physical signs were somewhat different from those which have been reported. The radiograms, however, made the diagnosis clear.

A boy, aged eight, was admitted into hospital on October 2nd, 1912, on account of sickness and vomiting of four days' duration.

He had always been healthy until his present illness, save for an attack of measles in infancy, and had seemed in good health on September 28th. Next morning he felt sick and vomited, and his mother kept him in bed. The vomiting recurred, and he became fevered, and was restless and rambled in his talk. The symptoms persisted, and on October 1st he complained of headache, and frequently put his hands up to his occiput.

On admission, he was mildly delirious and very restless, and constantly moved his limbs in an erratic way. He often placed his hands at the back of his head. The skin was pale, dry, and harsh. The face was flushed and the conjunctivæ were inflamed. He took his drinks well, but passed both urine and fæces in bed. There were few objective signs. There was a slight catarrh in the chest, and the tongue was dry and brown. The left membrana tympani showed some old cicatrices, and the right a large chronic perforation, from which issued a very small quantity of slightly odorous muco-pus; but there was no mastoid tenderness, and no palsy or spasm or inequality of reflex could be discovered, save that the right pupil was slightly larger than the left. Kernig's sign was negative. During the succeeding week slight improvement ensued, the delirium becoming less and the pulse rate diminishing in frequency. On October 5th, however, the stools contained a considerable amount of muco-pus, which persisted for twelve days. The pupillary inequality was inconstant. Râles were now audible at the bases of the lungs. On October 14th, however, he *suddenly* became extremely livid and very ill, and appeared to suffer abdominal pain. On October 17th the abdomen was for the first time somewhat distended, and a small consolidation was discovered at the base of the right lung. On the 20th new physical signs were recognized on the front of the chest. The percussion note at the left apex was skodaic in character, and an area of dullness ran outwards from the heart to the left shoulder; the respiratory murmur was very deficient (Fig. 252). The area of cardiac dullness was increased in every direction. The signs at the right base were unaltered. Little change was noticed during the next few days, but the dull area on the front of the chest had disappeared on the 26th, though the respiratory murmur at the left apex was still very deficient.

His general condition slowly improved, though the cerebral symp-

toms still persisted. On November 12th the chest was again examined, and the area of tympanitic percussion, shown in Fig. 253, was now recognized. It may have been present for a few days previously, as the chest was not examined on every day. The apex impulse persisted in the fifth interspace just within the nipple line. The consolidation at the right base was larger than it had been. Progress thereafter was continuous, though slow. The physical signs gradually cleared up, and on December 6th the front of the chest seemed normal. The liver was still enlarged and the basal consolidation was resolving. On dismissal, on January 19th, 1913, he was fat and well, though the pulse rate was still rather frequent. In June 1913 he was very well

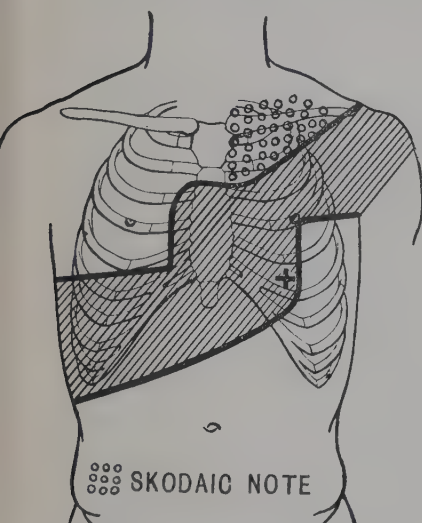


FIG. 252.—PNEUMO-PERICARDIUM.
Physical signs on October 20th.

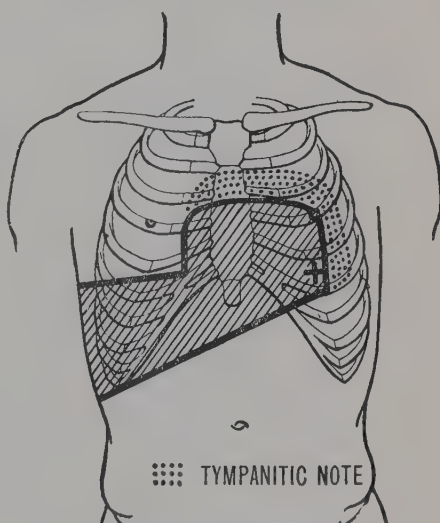


FIG. 253.—PNEUMO-PERICARDIUM.
Physical signs on November 12th.

and attending school regularly. There was still some dullness and deficiency of the respiratory murmur at the right base.

During his residence in hospital the fever was slight and only exceeded 100° F. on October 19th, 20th, and 22nd, reaching a maximum of 100·8° on the latter date. It never rose above 99° after December 16th. The pulse rate rose to 156 on October 3rd, and afterwards ran about 125 until the second week of December, when it tended to run at lower levels, but it was still in the neighbourhood of 100 on dismissal. The respirations numbered 44 on October 5th, and thereafter ran about 30, falling to normal limits in the middle of December.

The case presented many interesting features, and an accurate diagnosis of the sequence of events is impossible. On admission

it seemed probable that he was suffering from meningitis, but no source of infection was apparent; the aural sepsis seemed quiescent, and the course of events showed that the disturbance was general. The Widal tests, etc., were all negative, no sputum could be obtained, and a septicæmia of unknown nature was the only diagnosis. The cause of the exacerbation of the symptoms on October 14th was not recognized, and the pneumonia was not apparent until three days later. The interpretation of the new physical signs on October 20th was also difficult, and it was only on October 28th that his general condition permitted examination by the X-rays, and the pneumo-pericardium was recognized. The radiograms are instructive. On October 28th the distension of the pericardium was probably uniform, the heart was but little displaced, and the pneumonia was small (Fig. 254). On November 11th the distension of the sac was considerably larger, but only towards the left side and immediately below the apex, and the heart was displaced towards the right. On November 22nd the heart still seemed displaced, but the gas was in less amount and almost wholly on the left side (Fig. 255). On December 6th the distension was less and the heart had returned to its normal site. On December 16th the distension was still less, and subsequent radiograms failed to reveal any gas in the pericardial sac. The distension of the pericardial sac, however, was never quite uniform, for the apex impulse persisted throughout, and the area of cardiac dullness also remained, so that the heart can never have left the front of the chest. There was at no time any evidence of pericarditis, and there was no history of any illness in the past likely to have produced pericardial adhesion, so that the cause of the localization of the gas is unknown.

It seems possible that the original septicæmia provoked (or originated in) a local lesion of the mediastinal glands, which on October 14th became disseminated more widely, involving first of all the right lung and subsequently the pericardium and permitting the entrance of air into the pericardium from the lung.

The possibility of a gas-producing infection of the pericardium seems slight, for at no time was pericardial friction observed, and the radiograms exclude the presence of effusion.

The only radiogram which we can discover in the literature is that which was taken in Wenckebach's case. It differs in some respects from Figs. 254 and 255. The heart appears to

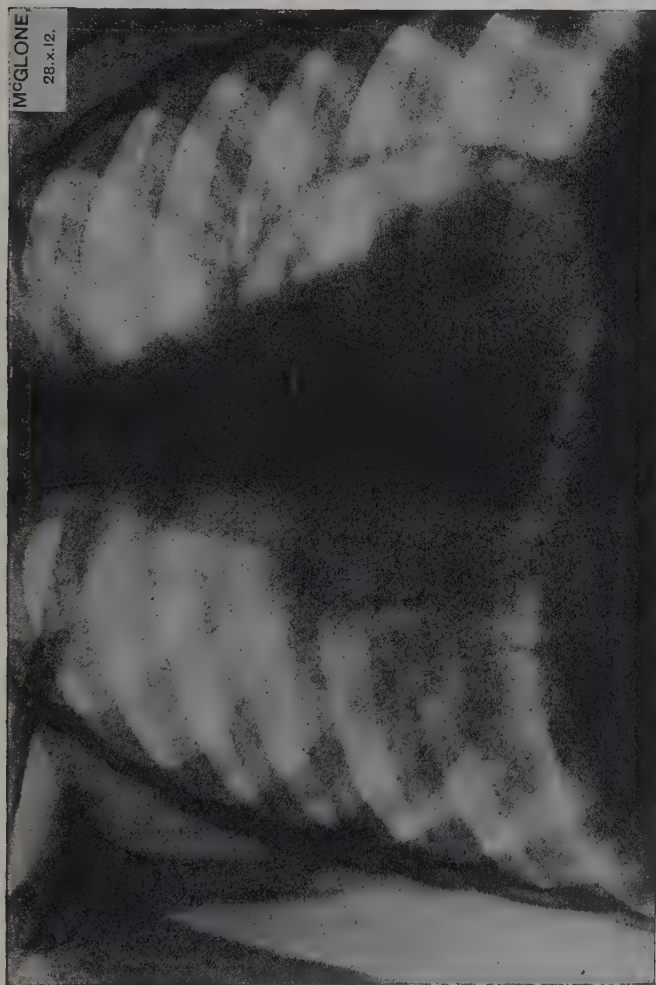


FIG. 254.—PNEUMO-PERICARDIUM (J. R. R.).
Skiagram taken on October 28th.

be in its usual situation, and is not displaced. The pericardial sac is uniformly distended and projects well to the right of the right auricle. The upper margin (as in Fig. 255) extends higher upon the left side of the chest than upon the right. The

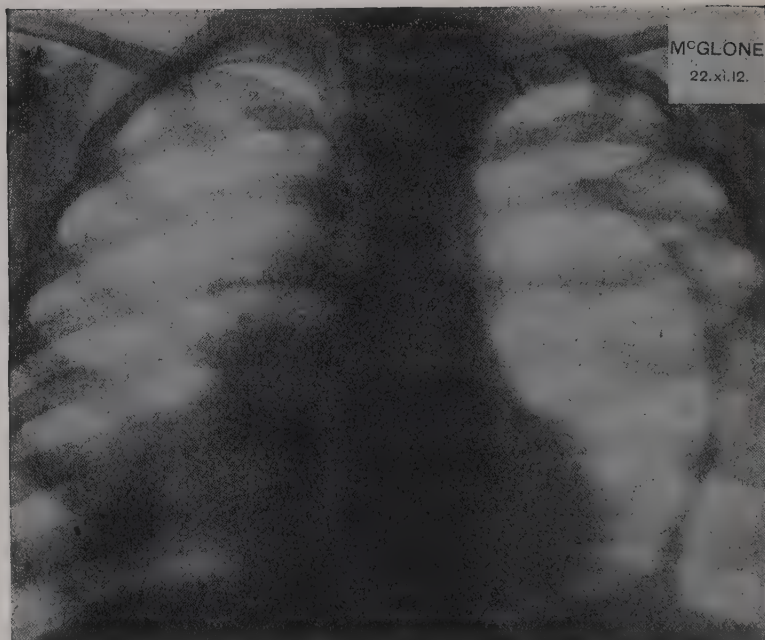


FIG. 255.—PNEUMO-PERICARDIUM (J. R. R.).

Radiogram taken on November 22nd.

lower border and the apex are not defined, as the heart in this situation dipped into the fluid effusion in the lower recesses of the sac.

CHAPTER XXVI

THE TREATMENT OF PERICARDITIS

THE treatment of acute pericarditis must be conducted on lines similar to those which have been considered with reference to acute endocarditis (Chapter XV).

The essential feature is *absolute rest*, both bodily and mental, so as to insure the minimal amount of cardiac strain, and a comfortable bed-rest is often required for patients who are unable to lie flat in bed. The restriction of active movements should be maintained until all the symptoms have disappeared, the pulse rate has returned to normal, and the area of cardiac dullness has reverted to its usual limits. The patient may then be allowed to move about in bed, but he should not be allowed out of it for at least two months longer, and for another month should be confined to a couch. Restrictions as to exercise must be continued for three to six months afterwards, and violent exercise prohibited for a year longer.

It has been suggested that physical exertion should be instituted early in cases of pericarditis, in order to augment the movements of the heart, and so limit the area of adhesion ; but if, as we believe is the case, one of the main functions of the pericardium is to prevent undue distension of the heart during physical exertion, early activity is likely to distend the inflamed sac and so prevent it from exercising this particular function. It is not uncommon to see the right heart dilate temporarily from even trivial exertion early in convalescence, and the patients in whom the period of rest had been prolonged have always seemed to us to have hearts smaller than those in whom physical exertion had been rapidly resumed. Adhesion of the visceral and parietal layers seems to interfere little with the cardiac action, unless mediastinitis co-exists and the heart is fixed to the chest wall, or the pericardial sac is unduly large.

Pain when present must be relieved, in particular in those cases in which it interferes with sleep. Local applications, either hot or cold (an ice poultice is generally comforting) or sedative applications, such as glycerine of belladonna, may be sufficient; leeches or blisters are sometimes successful; but opium is often needed. Pulv. ipecac. co., or a hypodermic injection of morphia, is generally useful.

In many cases no particular line of treatment is indicated beyond that instituted on account of the causal condition. In acute rheumatism salicylate treatment should be continued, but large doses (above 120 grains *per diem*) should be avoided. In cases where cardiac failure is impending stimulants may be required. Ammonia, nux vomica, and alcohol have seemed to us to be of special value in addition to drugs of the digitalis group.

In cases with effusion the question of performing paracentesis pericardii naturally arises, but the general impression that it is rarely required is probably correct, as death is seldom due solely to the pressure exercised upon the heart. The results of paracentesis, too, are by no means favourable. In West's series 33 out of 67 cases died, including 4 out of 11 rheumatic cases; and in Delorme and Mignon's series 65 per cent. of 82 cases proved fatal. The results of incision and drainage are also unfavourable, though in a few cases recovery has ensued in apparently moribund patients. In our series no operation was performed, though in one case it seemed desirable. The patient was the boy from whom the radiogram (Fig. 245) was obtained, and the pericarditis, which was sero-fibrinous, was secondary to a septic broncho-pneumonia, accompanied by left pneumothorax. Paracentesis seemed impossible, and the boy's general condition from the pulmonary aspect negatived operative interference of a more extensive kind. In another case *post-mortem* examination indicated the desirability of the operation. The apex of the heart was, however, firmly adherent to the parietal pericardium and to the chest wall, and had remained immobile and well marked during the whole progress of the illness, so that the degree of the effusion was not estimated correctly, the main factor in the increase of the area of cardiac dullness being ascribed to dilatation. Six of the 30 cases recovered; but in none of

the remaining 22 did the pericarditis appear chiefly responsible for the fatal issue.

In purulent pericarditis incision and drainage seem indicated. We have never seen a case either during life or on the *post-mortem* table.

Many sites have been suggested for the operation, but the most suitable seems that suggested by Baizeau and Delorme and Mignon—namely, puncture as near as is possible to the sternal margin in the fifth or sixth left intercostal space. The internal mammary vessels and the left pleural cavity are thus avoided. The presence of friction at this point, or loud cardiac sounds showing that the heart is immediately underneath, contra-indicate this site, and puncture may then be performed from below upwards through the left costo-sternal angle, or just inside the left border of dullness and outside the cardiac impulse. The pleural cavity is necessarily opened in the latter case.

The treatment of cases of adherent pericardium in which symptoms of cardiac insufficiency are present must be conducted upon lines similar to those which are of value in chronic valvular disease. Fibrolysin has been recommended in those cases with a view to the dissolution of the adhesions, but there are no records of any very notable success. In one of our cases the patient seemed slightly better after its use.

Operative treatment has been advocated in those cases of adherent pericardium where the heart is firmly fixed to the thoracic wall, resection of those portions of the ribs, costal cartilages, or sternum which are involved relieving the heart from much unnecessary work. A widespread and forcible movement of the bony and cartilaginous chest wall is the special indication for the operation. In many cases of adherent pericardium, however, the cardiac difficulties arise in other ways from adhesion to the diaphragm, lungs, or mediastinum, or from cicatricial bands around the heart or the great vessels; and suitable cases for operation are by no means frequently seen. The dangers of the operation are considerable, sudden death on the operating-table (which occurred in the only case with which we have been personally concerned) being not infrequent, and pleurisy often ensues, as the pleural cavity is almost necessarily opened during the operation. We believe,

however, that a more careful selection of cases and an earlier operation than has hitherto been carried out, will in the future lead to better results.

A similar operation has been suggested in cases of cardiac disease accompanied by much enlargement of the heart in *adult* patients. The contour of the chest wall is rarely deformed in adults by cardiac hypertrophy, as the rib arches are unyielding, and the increased space within the thorax demanded by the heart can only be supplied by depression of the diaphragm and lessening of the pulmonary area. The removal of portions of the ribs permits enlargement of the heart forwards and so increases the pulmonary area. The results which have been obtained are at any rate encouraging.

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CHAPTER XXVII

CONGENITAL HEART DISEASE

THE DEVELOPMENT OF THE HEART

THE primitive heart, formed by coalescence of the caudal portions of the two primitive ventral aortæ, is a simple tube which soon becomes differentiated into its component parts and bent on its long axis, thereby becoming S-shaped with its posterior, venous, end above and behind the anterior, arterial, end. Posteriorly the veins open into the sinus venosus, which, communicating by a single lateral aperture with the auricle, is connected with the ventricle by means of a short, narrow segment, the auricular canal. The ventricle opens anteriorly into the bulbus cordis, from which the primitive aorta, the truncus arteriosus, arises.

The subsequent enlargement of the heart is effected mainly by further development of muscle fibres in the auricle and ventricle. The tissue representing the sinus venosus and auricular canal retains its special characters and, constituting the primitive tissue of the adult heart, is found only at certain sites. In the frog, the turtle, and other lower vertebrates, the auricular canal persists. But in the mammalian heart the auricular canal is represented only by the auriculo-ventricular node, the bundle, and its branches (*see* p. 5); the sinus venosus is represented mainly by the sinus node, and to a lesser extent by other remnants of primitive tissue submerged in the auricular muscle at the mouth of the coronary sinus and elsewhere in the auricular wall. The bulbus cordis becomes incorporated in the conus arteriosus of the right ventricle.

In the fourth week the division of the single heart into two halves, right and left, begins by the growth of three septa.

The Inter-auricular Septum is formed by the fusion of two septa. In the fourth week the septum primum begins to grow

downwards from the roof of the auricle, and as the anterior and posterior parts grow more rapidly than the central portion, the septum at one stage of its development is sickle-shaped, there being a large aperture, the ostium primum, in its lower part, which still affords communication between the right and left auricles. Soon the ostium primum is closed, and simultaneously the upper part of the septum becomes perforated, thus forming the ostium secundum. The second auricular septum grows downwards immediately to the right of the septum primum and fuses with it, leaving however an aperture, the foramen ovale, which does not coincide accurately with that in the septum primum. The foramen ovale is therefore valved and permits, during foetal life, free communication between right and left auricles. The foramen ovale usually closes during the first weeks after birth, but in many adults—in 30 per cent. according to Shennan¹—there is inoclusion, the foramen persisting as a narrow valved channel between the auricles.

The Inter-ventricular septum appears in the fourth week. It grows upwards and backwards from the apex of the single ventricle, and is at first crescentic with its concavity upwards. The anterior portion ultimately fuses with the left side of the truncus arteriosus; the posterior part unites with the septum dividing the auricular canal; the median portion, joining the septum of the truncus arteriosus, forms the membranous septum of the ventricles. The inter-ventricular septum is completed towards the end of the sixth or seventh week, a few days after the septum of the truncus arteriosus is completed.

The Septum of the Truncus Arteriosus, dividing this vessel into aorta and pulmonary artery, appears in the fifth week. It develops from above, sagittally, and in its downward growth undergoes rotation towards the right. Thus the left trunk, the pulmonary artery, becomes anterior. As already stated, this septum unites at its lower end with the median portion of the inter-ventricular septum. Abnormal rotation of the septum of the truncus arteriosus may result in transposition of the aorta and pulmonary artery, the former arising from the right, the latter from the left, ventricle. Incomplete fusion of the septum of the truncus arteriosus with the median

¹ Shennan, T., *Post Mortems and Morbid Anatomy*, Lond., 1912, 60.

portion of the inter-ventricular septum may also occur. The ventricular cavities then open into each other, while the arterial trunks arise from one or both ventricles as the case may be.

Of the primitive aortic arches, the fourth right becomes the right subclavian artery ; the fourth left, the aorta ; the sixth right arch becomes the right pulmonary artery ; the distal portion of the sixth left arch forms the ductus arteriosus.

The Etiology of congenital anomalies of the heart has afforded much discussion. The deformity may represent merely the developmental adjustment of an otherwise normal heart to mechanical strain, as in ectopia viscerum.¹ Regarding the chief developmental anomalies, however, the problem is whether they are due to endocarditis in foetal life, as was advocated by Lancereaux, or to arrest of development as was maintained by Rokitansky. Two or more anomalies often co-exist. One of the most frequent anomalies is pulmonary stenosis accompanied by a defect of the inter-auricular, or inter-ventricular, septum ; and the problem under discussion has been whether the pulmonary stenosis was primary and due to foetal endocarditis, the septal defect being secondary thereto and in part compensatory, or whether both anomalies are due to arrest of development. Among the weighty arguments against the acceptance of Lancereaux's theory is the fact that the septum of the truncus arteriosus and the inter-ventricular septum are both complete within a few days of each other ; consequently if pulmonary stenosis is due to endocarditis this must occur within one particular fortnight, namely the seventh and eighth weeks. Moreover, from the close proximity of the pulmonary to the aortic orifice, an inflammation affecting the former so severely as to cause its stenosis would almost necessarily affect the aortic orifice also ; whereas the latter orifice is rarely affected. Further arguments in support of the theory of arrested development are that a stenosis of inflammatory origin necessitates a considerable time for its development, and that microscopic examination usually fails to reveal any evidence of inflammation.

The theory of arrest of development receives very convincing

¹ Robertson, J. I., *Journ. of Path. and Bacteriol.*, 1913, xviii., 211.

support from the observations of Keith,¹ who has shown that pulmonary stenosis, one of the most frequent of congenital anomalies, and one which by raising the pressure on the right side of the heart tends to prevent closure of the inter-auricular and inter-ventricular septa, is due to arrest of growth of the bulbus cordis. It would lead us beyond the scope of this work to speculate whether the fundamental cause of the arrest of development is due to the extrusion, from the primitive germ cells, before they became gametes, of polar bodies which had in them determinants for the development of the bulbus cordis, the septa, or other parts of the heart. Evidence in favour of this hypothesis will be found in Berry Hart's *Phases of Evolution and Heredity* (1910). It is, however, well known that acute endocarditis is very prone to arise at the site of a congenital defect, which may then become so disguised that its real nature is not appreciated. Thus a congenital anomaly may be the fundamental etiological factor of a valvular lesion which arises without any adequate, or apparent, cause in post-natal life.

In the following pages we shall consider the congenital anomalies which are of clinical significance and not deal with ectopia cordis and other malformations which are mainly of teratological interest.

DEXTROCARDIA

In true dextrocardia, transposition of the heart, electrocardiograms by Derivation *I* show inversion of the deflexions *P*, *R*, and *T*, but are otherwise normal.² The other viscera are usually transposed too ; and these cases of complete transposition are almost invariably unattended by any symptoms. Dextrocardia without transposition of other viscera is usually associated with other cardiac abnormalities, such as transposition of the great vessels or patency of the septa, and is therefore more often associated with symptoms.

DEFECTS OF THE INTER-AURICULAR SEPTUM

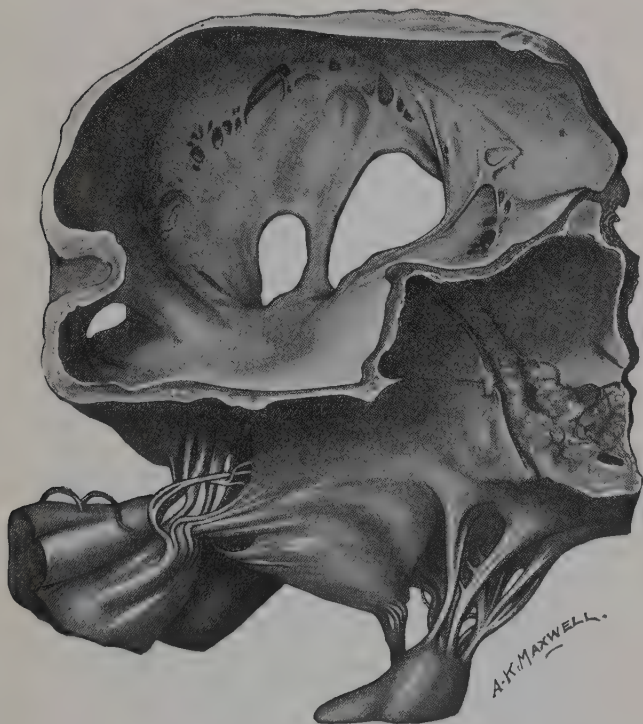
Entire absence of the inter-auricular septum is very rare, but the condition is not incompatible with the attainment of

¹ Keith, A., *Studies in Pathology*, Aberdeen, 1906, 55.

² Willius, F. A., *Amer. Journ. Med. Sci.*, 1919, clvii., 485.

adult life, and the individuals are not necessarily cyanosed.

Partial defects of the inter-auricular septum are probably the most frequent of all congenital anomalies. They occurred in 182 of Abbott's¹ series of 412 cases of congenital heart disease.



Defect of Inter-auricular Septum.

FIG. 256.—PORTION OF HEART OF BREWER'S DRAYMAN BETWEEN THIRTY AND FORTY YEARS OF AGE, WHO FELL DOWN IN THE STREET AND EXPIRED SUDDENLY.

Two large apertures at the anterior part of the inter-auricular septum.
(From a specimen in the Museum of the Royal College of Surgeons of Edinburgh).

Persistent Ostium Primum, due to arrested development of the septum primum, occurs as an aperture of variable size and of circular or crescentic form, in the lowest part of the

¹ Abbott, M. E., Osler and McCrae's *System of Med.*, Lond., 1908, iv., 338, 348.

inter-auricular septum (Fig. 256). The anterior segment of the mitral, or the medial cusp of the tricuspid, valve is usually cleft or otherwise deformed.

Patent Foramen Ovale is more frequent. Under this term we do not include a valved foramen ovale such as may be found in about thirty per cent. of all hearts *post-mortem*, but refer to an orifice which, representing inocclusion of the ostium secundum (see p. 454), permits free and constant communication between the two auricles. This defect may co-exist with a patent ostium primum. Either ostium, or both, may remain patent if there is arrested development of the bulbus cordis, and the latter is then partly compensated by the septal defect.

Partial defects of the inter-auricular septum do not invariably give rise to symptoms or physical signs, and are not incompatible with long life. One of Blackhall Morison's¹ cases, a woman of forty-three years, was the mother of five healthy children, yet the persistent ostium primum admitted the forefinger; another was a man who died at the age of seventy-two, having a stenosed pulmonary orifice and a large patent foramen ovale. Auricular septal defects often produce no physical signs. In some instances a murmur, systolic or pre-systolic in time, is heard over the præcordia, and is usually loudest over the base. The murmur, which is seldom accompanied by a palpable thrill, may be audible posteriorly, but it is not conducted into the carotids. A girl of eight years, shown to one of us by Dr. J. G. Cattnach, probably suffered from this malformation. She was well developed, and presented no cyanosis, dropsy, or clubbing of the fingers, but had been subject to a cough for some years. The cardiac impulse was not visible, and no thrill was palpable; but a loud blowing systolic murmur, heard all over the præcordia, was maximal behind the right half of the sternum at the level of the fourth intercostal space. It was not conducted into the neck, nor towards the left clavicle.

The recognition of the septal defect is often rendered difficult by co-existing incompetence of the mitral, or tricuspid, valve.

¹ Blackhall-Morison, A., *Journ. of Anat.*, 1919, liv., 90.

DEFECTS OF THE INTER-VENTRICULAR SEPTUM

These were noted in 161 of Abbott's series of 412 cases of congenital heart disease. Complete absence of the inter-ventricular septum (Fig. 257) is a very rare form of tricælian

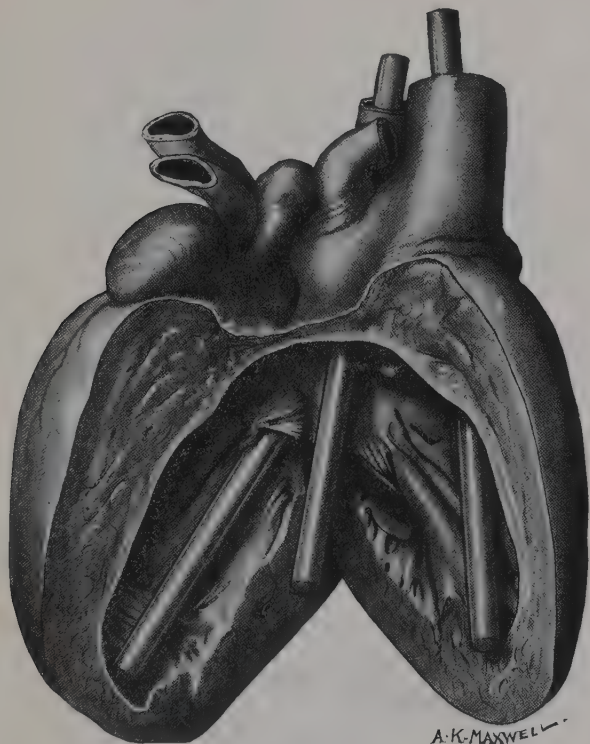


FIG. 257.—TRICÆLIAN HEART OF A CHILD.

The single ventricle is laid open. The rods are placed from left to right respectively, in the tricuspid orifice, the pulmonary artery, and the aorta.

(From a specimen in the Museum of the Royal College of Surgeons of Edinburgh.)

heart, but the patient may attain adult life without cyanosis or other symptoms arising, despite the admixture of arterial and venous blood.

A Partial Defect is much more frequent. It is usually at the basal part of the septum, and may involve the membranous

septum (Fig. 258). Other developmental defects, such as pulmonary stenosis or atresia, often co-exist. For example, in the heart of a child two years of age, the conus arteriosus was small, indicating arrested development of the bulbus cordis, and there was an aperture, 1.2 cm. in diameter, at the base of the septum below the aortic valve.¹

The murmur (bruit de Roger) occasioned by a partial defect of the inter-ventricular septum is usually loud and harsh. It commences early in systole, and is prolonged into diastole, thus obscuring the second sound, which may, however, be heard distinctly at the base. The murmur has its site of maximal intensity at the left side of the sternum close to the xiphoid, and is not well conducted into the carotid artery or into the axilla. This murmur was heard in an engineman who died at the age of forty-four, and in whom there was found, at the anterior angle of the membranous septum, a conical aperture about one-eighth of an inch in diameter, surrounded by cicatricial tissue and surmounted by recent vegetations.²

A septal defect may involve the bundle and thus produce auriculo-ventricular block. Of 21 cases of auriculo-ventricular block in children, nearly all were definitely or probably of congenital origin, the only exceptions occurring during severe and usually fatal diphtheria.³

PULMONARY STENOSIS

This anomaly, which was shown by Keith⁴ to be due to arrested development of the bulbus cordis, is not infrequent. Not only is the pulmonary artery narrow, but the conus arteriosus is usually small, while the right ventricle as a whole is enlarged and its wall hypertrophied (Fig. 258). The pulmonary valve may be deformed and be represented by a diaphragm or a tapering funnel. Defects of the inter-auricular, or inter-ventricular, septum, and patency of the ductus arteriosus, often co-exist as a result of the hindrance to the outflow of

¹ Cowan, J. M., and Ferguson, A. R., *Lancet*, 1903, ii., 952.

² Cowan, J., and Storey, L., *Glasgow Med. Journ.*, 1909, lxxii., 425.

³ Eyster, J. A. E., and Middleton, W. S., *Amer. Journ. Dis. of Children*, 1920, xix., 131.

⁴ Keith, A., *Studies in Pathology*, Aberdeen, 1906, 55.

blood from the right ventricle, and these secondary defects are in some degree compensatory.

The condition is usually detected in infants or young children. Cyanosis is the rule. Although this sign may be con-

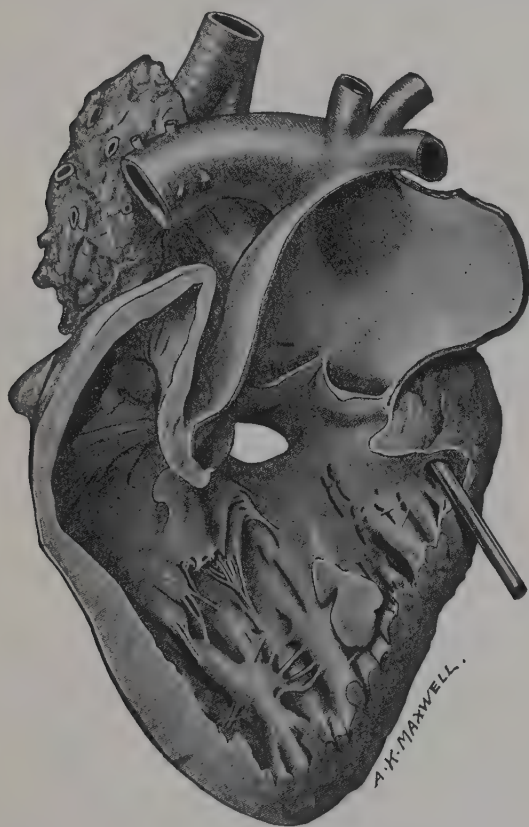


FIG. 258.—HEART OF A CHILD SHOWING IMPERFECT INTER-VENTRICULAR SEPTUM.

The ascending part of the aorta, which arises from both ventricles, is laid open. A stilette passes into the conus arteriosus, the diameter of which is 0.3 cm.

(From a specimen in the Museum of the Royal College of Surgeons of Edinburgh.)

spicuous only when the child is coughing or crying, it is in this congenital anomaly that pronounced and persistent cyanosis is most common. It is most severe when there is atresia

of the pulmonary artery, but even in stenosis a purple colour of the face, clubbing of the fingers (Fig. 260), a high degree of polycythæmia in which the number of red corpuscles may amount to over nine millions per c. mm., dyspnœa, and undue susceptibility to cold are usually observed.

The chief physical signs are a systolic thrill in the second, or second and third, left intercostal spaces close to the sternum, a harsh systolic murmur with enfeeblement or absence of the second sound at the same area (Fig. 259), and evidence of hypertrophy of the right ventricle. In one of our cases, a girl aged eleven years, in whom there was also a partial defect of

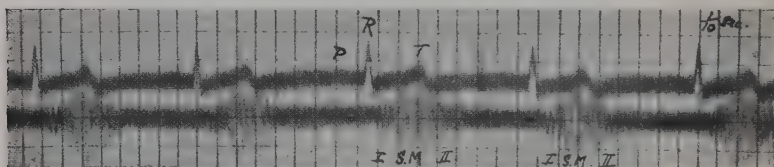


FIG. 259.—ELECTROCARDIOGRAM (DERIVATION II) AND PHONO-CARDIOGRAM FROM SECOND INTERCOSTAL SPACE.

Congenital pulmonary stenosis. I, first sound; SM, systolic murmur; II, second sound.

(H. L. Watson-Wemyss and J. D. Gunn, *Edin. Med. Journ.*, 1913.)

the inter-ventricular septum, the electrocardiographic signs of right ventricular preponderance were well marked. In some instances the physical signs point also to incompetence of the pulmonary valve. Thus a girl, three and a half years of age, in whom there was no evidence of any serious antecedent illness, was stunted in growth but not cyanotic, although the superficial veins of the forehead and temporal regions were dilated, and the jugular veins turgid. The cardiac impulse was forcible from the left margin of the sternum outwards to the cardiac apex; a systolic thrill was felt in the pulmonary area; and loud systolic and diastolic murmurs were audible in the same area.

Infants with pulmonary atresia seldom survive for more than a few months. A few of those with pulmonary stenosis attain adult life, but most of them die in childhood or about the time of puberty, frequently from pulmonary tuberculosis.

PATENT DUCTUS ARTERIOSUS

Patency of the ductus arteriosus is, in our clinical experience, the most frequent congenital anomaly, occurring in nine of twenty-one cases. In Abbott's series of 412 cases of congenital disease, there were 111 of patent ductus ; only two other anomalies were of greater frequency, namely defects of the inter-auricular septum (182 cases) and defects of the inter-ventricular septum (161 cases).



FIG. 260.—CLUBBING OF FINGERS.

In the ante-natal state, venous blood issuing from the right ventricle by the pulmonary artery is carried through the ductus arteriosus into the aorta, whereby it is conveyed to the placenta and there oxygenated. Until birth the diameter of the ductus arteriosus is greater than that of the pulmonary arteries ; but by the eighth day after birth, the ductus is practically closed,

and all the blood discharged from the right ventricle passes through the pulmonary circulation. In rare instances the ductus remains patent, usually for the purpose of compensating some other congenital anomaly of the heart. The two chief causes of persistent patency of the ductus arteriosus are (1) stenosis or atresia at or near the orifice of the pulmonary artery, in order that the pulmonary branches may be supplied with blood from the aorta ; and (2) stenosis of the aortic orifice, so that the aorta may be filled from the pulmonary artery. Thus in some instances the flow of blood is from the aorta to the pulmonary artery ; in others from the pulmonary artery to the aorta.

Although usually associated with other congenital anomalies, a patent ductus may be the most conspicuous, and indeed is often the only, anomaly recognizable clinically. The condition is observed more often in the male sex, and may be detected not only in infancy and childhood, but also in individuals well up in years. There are often no symptoms, and the anomaly may not be recognized until the child is examined during the course of an ailment such as bronchitis or measles, or until, in later life, he presents himself for military service or for life insurance. In these individuals there is but little interference with the circulation of the blood, the lumen of the ductus is small, and clinically no other cardiac anomaly can be detected.

These individuals complain of no symptoms referable to the heart, they are not cyanotic, have no clubbing of the fingers or toes, and may be capable of undertaking strenuous physical exertion. One of our patients, aged twenty-three, had over three years' active service in the Army, including two years in Gallipoli and in Egypt, before he reported sick with bronchitis. Another, a maid aged thirty-two, has never presented any evidence of cardiac distress.

The most conspicuous, and often the sole, sign of the anomaly is the characteristic murmur. It is loud, harsh, or blowing in character, and is best heard in the second left intercostal space, about two inches from the sternum, where a systolic thrill can generally be felt. The murmur may be wholly systolic in time, but is most characteristic when, beginning distinctly after the commencement of the first sound, it accompanies the latter part of that sound, occupies the short pause,

accompanies the second sound and finally dies away during the long pause.¹ The murmur is usually loudest at its commencement and diminuendo, but it may wax about its middle and then wane. The second sound is usually distinctly audible.

The murmur is well conducted towards the left clavicle but is not conducted downwards, to the right, or into the carotid arteries. It may, however, be heard best in the left inter-scapular region, and then is probably produced by blood flowing from pulmonary artery into aorta, the vibrations being loudest where the junction of blood-currents is nearest to the surface. Aortic or pulmonary stenosis may give a special emphasis at a particular site to the first portion of the sound. In some instances the murmur heard posteriorly waxes during inspiration and wanes during expiration, and the arterial pulse may then be paradoxical, namely of smaller volume during inspiration than during expiration, because the fall of intrathoracic pressure during inspiration leads to aspiration of blood from the pulmonary artery and a consequent increased flow of aortic blood through the ductus.

In one of our cases, that of a boy four years of age, without any history of antecedent acute infective disease, the indubitable signs of patent ductus arteriosus were discovered when the otherwise perfectly healthy child developed a coryza. The murmur was conducted outwards to the left shoulder and was audible in the left inter-scapular region. Six years later, when we again examined the boy, he was apparently in good health but the physical signs were unchanged. A point of some interest is that in the boy's paternal grandfather a congenital anomaly of the heart had been detected many years previously by the late Dr. George Balfour.

In another case, a loud, basal, systolic murmur was detected in an infant soon after birth. When five years old he was a bright intelligent child, active and well developed, without a trace of cyanosis even on coughing or crying, and without any clubbing of the fingers or toes. The heart was not enlarged, the sounds at the apex were pure and clear, there was no increase of dullness at the base of the heart, nor was any pulsation seen in that region. But in the second and third left intercostal spaces near the sternum a systolic thrill was felt, and a loud, harsh, systolic murmur was audible. This was conducted upwards and outwards to the mid-point of the left clavicle, and was also audible in the left inter-scapular region. The second sound was pure and distinct.

When the interference with the circulation is of moderate

¹ Gibson, G. A., *Edin. Med. Journ.*, 1900, N.S., viii., 1.

degree, yet is so well compensated that the individual complains of no symptoms, physical signs indicating enlargement of the right ventricle, and especially of the conus arteriosus, and dilatation of the pulmonary artery, are superadded to the auscultatory signs already mentioned. An impulse, synchronous with that of the cardiac apex, may then be felt in the second and third left intercostal spaces near the left margin of the sternum. The cardiac dullness in the same region may extend unduly to the left of the sternal border, as was pointed out by Gerhardt. This area of dullness was observed in one of our cases, a well-developed, athletic man of twenty years, who had suffered from no serious illness except diphtheria at the age of four, and who presented a loud murmur, unaccompanied by a thrill, but characteristic in its point of maximal intensity and in its conduction towards the left clavicle.

The enlargement of the conus arteriosus may be obvious by radioscopy, or in a radiogram, the shadow of the pulmonary curve, which pulsates freely, being large and bulging prominently outwards towards the left. In exceptional cases the patent ductus is the seat of an aneurysmal dilatation. In only one instance have we observed distinct electrocardiographic evidence of right ventricular preponderance; in all our other cases the electrocardiograms were of normal form.

If the defect is more pronounced, the child may be small for his age, or even stunted in growth, a little dyspnoic on exertion, and slightly cyanotic on crying or on coughing. These signs were observed in a boy, eleven years of age, in whom the fingers were slightly clubbed. At the inner end of the second and third left intercostal spaces, there was a visible and palpable impulse, systolic in time, and a purring systolic thrill. A harsh systolic murmur, maximal at the third left costal cartilage, was not conducted downwards, nor to the left, nor into the vessels of the neck, but was well heard on the left side of the vertebral column from the level of the spine of the scapula down to the tenth dorsal vertebra. This murmur, posteriorly was louder during inspiration than during expiration, and the radial pulse was paradoxical during deep respiration. In this case the patency of the ductus was probably secondary to pulmonary stenosis. A year later the patient died of pulmonary tuberculosis.

TRICUSPID, AND MITRAL, STENOSIS AND INCOMPETENCE

Congenital anomalies of the tricuspid valve are not frequent, and those of the mitral valve are even more rare. The physical signs are described in Chapter XVIII, but it must be remembered that the valvular lesion is often associated with other defects. One of our cases of tricuspid stenosis and incompetence, a boy sixteen years of age, who was also suffering from tuberculous peritonitis, was deeply cyanosed even when resting; he was stunted in growth, and the fingers were clubbed. A systolic thrill was felt all over the enlarged heart, and loud systolic and diastolic murmurs were audible, being maximal over the fourth left chondro-sternal articulation; the liver was felt to pulsate forcibly, and the superficial veins in the temporal regions were seen to pulsate freely.

Diagnosis of Congenital Heart Disease

In infants, the association of cyanosis and a loud, harsh murmur, with or without a præcordial thrill, affords a sure basis for the recognition of a congenital anomaly. In children and in adults, the diagnosis rests mainly on the age at which the murmur was first detected, on the absence of any history of chorea or other rheumatic infection, and on the significance of the physical signs. In illustration of acquired cardiac disease simulating congenital anomalies we recall a boy nine years of age, in whom a loud diastolic aortic murmur was audible. He was not cyanosed; the fingers were not clubbed; he had suffered from interstitial keratitis, and, when aged eight, from syphilitic arthritis of the knees. The cardiac lesion was obviously another manifestation of congenital syphilis, and post-natal in its development. Another case was that of a girl aged four years, well developed, without cyanosis, thrill, or cardiac enlargement, in whom a loud systolic murmur was heard. It was maximal just above and within the left nipple, but was not transmitted into the axilla, nor upwards, nor to the right. The murmur was considered to be evidence of mitral incompetence as a sequel to two short, sharp, febrile illnesses of indefinite nature, a few months previously.

Septal defects are not necessarily attended by any cyanosis; the murmurs they occasion do not correspond in point of maximum intensity with, and are not transmitted in the same

manner as, those of valvular lesions. The murmurs of patent ductus arteriosus and of defect of the inter-ventricular septum resemble each other in one respect, namely that they may occupy both systole and diastole without any interval between the systolic and diastolic portions. Such murmurs cannot originate at any of the valves, and occur only when a communication exists between separate cavities or vessels. Similar murmurs may be heard in aneurysmal varix as well as in ulcerative endocarditis or aneurysm, if rupture takes place between adjacent cavities or vessels.

The most extreme grade of cyanosis is observed in pulmonary atresia and stenosis. The recognition of a patent ductus arteriosus is relatively easy. Even in the entire absence of symptoms of cardiac origin, of cyanosis, and of clubbing of the fingers, the characteristic murmur in the second left inter-costal space, with or without a systolic impulse and thrill, can hardly be mistaken. If the murmur is associated with these additional signs and with evidence of hypertrophy of the dextral chambers of the heart, pulmonary stenosis probably co-exists.

The form of the heart, as seen by radioscopy or radiography, presents no characteristic change except in patent ductus arteriosus, as described on page 466. In cases of defect of the inter-ventricular septum, however, the right auricle may pulsate vigorously, synchronously with the ventricle, probably because the tricuspid valve is incompetent.

The Prognosis is relatively good if there be no cyanosis, clubbing of the fingers, or other signs of hindrance to the circulation. If the only defect is a septal one the prognosis is less unfavourable than in cases of pulmonary or tricuspid stenosis ; while the prognosis of uncomplicated patent ductus arteriosus is, on the whole, good.

The Treatment of patients affected with congenital heart disease resolves itself into the promotion of the general health, the avoidance of undue strain on the heart, and the prevention as far as possible of infection and particularly of pulmonary tuberculosis, which is in many cases the immediate cause of death. The risks are greatest at puberty and in early adult life.

CHAPTER XXVIII

THE DISEASES OF THE ARTERIES

1. **Arterio-Sclerosis.**—A diffuse affection involving the whole arterial tree.

Caused by HIGH BLOOD-PRESSURE :

- (a) Associated with renal disease.
- (b) From “repletion,” relative or absolute.
- (c) From intoxications due to—
 - (1) Metabolic errors ; gout, digestive disturbances, etc.
 - (2) Poisons introduced from without—lead, nicotine, etc.
- (d) Following the infections.
- (e) In polycythæmia.

2. **Focal Lesions.**—A patchy affection involving small portions of the arteries.

- (a) **INFECTIVE LESIONS :** due to staphylococci, streptococci, *B. anthracis*, *B. tuberculosis*, *Spirochæta pallida*, etc.

- (b) **WITHOUT DEFINITE EVIDENCE OF INFECTION :**

- (1) *Atheroma*, caused by—
 - (i) The infections : Enteric fever, tuberculosis, diphtheria, smallpox, measles, scarlatina, erysipelas, pneumonia, acute rheumatism, influenza.
 - (ii) Intermittent elevation of the blood-pressure.
- (2) *Mesarteritis*, caused by—
 - Syphilis, acute rheumatism, enteric fever, diphtheria.

- (3) *Endarteritis obliterans*, caused by—
 - (i) Syphilis, tuberculosis, acute rheumatism, diphtheria, scarlatina, small-pox, enteric fever.
 - (ii) Occlusion of the lumen from whatever cause.
- (4) *Medial Calcification*, caused by—
 - (i) Trauma (excessive use of limbs, intermittent or continuous elevation of the blood-pressure).
 - (ii) Adrenalin, nicotine, digitalin, hydrastine, barium chloride.

It has been customary to consider the arterial wall as composed of three layers, the intima, the media, and the adventitia, but while this is useful from the descriptive point of view, it tends to produce the idea that, physiologically, the layers are distinct and separate, an idea which is inaccurate. The internal elastic lamina, it is true, in the smaller vessels abruptly marks the line of division between intima and media, but it is absent in the aorta and larger vessels where the precise limits of the layers are indefinite. Muscular fibres are not confined to the media, but are also present in the intima and the adventitia; and although they are, in the smaller vessels, the chief constituent of the middle layer, they are relatively scanty in the larger trunks where the elastic tissue is the essential element. The two groups of vessels must be sharply separated. One group are the "mains" for the supply of blood to the tissues, with a calibre proportionate to the amount of blood which they have to accommodate, and a fibrous elastic structure adapted to resist the varying degrees of blood-pressure to which they are exposed. The other group are the "supply pipes," with muscular "taps" which can be partially closed, and so arrange the distribution of the blood to the various organs. The adventitia is but a part of the general connective tissue, and can seldom be exactly demarcated, and the connective and elastic frameworks of the three layers are structurally in contact. The essential element of the vessels is, as the late Professor Coats maintained, the intimal pavement endothelium.

The proportions of the three layers may vary even in the

same vessel in different parts of its course. Particular places may be exposed to special stress, and the liability to local damage is to a certain extent compensated. The upper wall of the ascending aorta, exposed to the full force of ventricular systole, is much thicker than the under side where the systolic impact is less direct. The vessels, too, are always thickened at bifurcations and around the origin of branches to meet the extra strain. Arteries such as the facial, superior mesenteric, and brachial, are exposed to injury from the movements of the neighbouring parts, and possess, in consequence, an adventitia which is notably thick. Others, such as the aorta and the nutrient vessels of the bones, are protected by their situation from such influences, and have an adventitia of little strength. The relative proportions of the different elements of each layer vary in vessels of similar size. The media of the renal artery, for instance, contains a much larger amount of muscle and a much smaller amount of elastic tissue than such a vessel as the carotid. One cannot predicate what the exact constitution of an artery will be, since the individual elements may vary in amount from its special function or its special site.

The vascular arrangements of the arterial walls point the same moral. The nutrition of the vessel is maintained by two sources. The vasa of the adventitia penetrate for a certain distance into the media of the larger vessels, though under normal conditions they do not trespass far. The minute vessels have no vasa and must be largely dependent upon the blood within them, although no definite lymphatic connection is apparent. It is difficult to estimate how much of the wall is supplied from each source, but in endarteritis the cells nearest to the blood-stream are always less degenerate than those farther away. In some cases of medial fibrosis the innermost muscle cells persist, while those in the centre of the layer have disappeared (Fig. 263). So it seems clear that in abnormal circumstances both the media and the intima may be nourished by the intra-arterial blood. The muscle of the media, however, may be intact in cases of endarteritis, and the vasa are sometimes seen to penetrate into the intima, so that both layers may at times be supplied from without. It seems probable that in normal conditions the medial supply is chiefly

vasal, and the intimal supply mainly intra-arterial, though in abnormal states a defect in one supply can be more or less compensated by the other. New formed vessels in the media or intima are, however, always branches of the vasa.

The lymphatic flow takes place from within outwards, though no definite lymph channels have been found in the vessel wall. But in early examples of endarteritis the muscle cells in the vicinity of the degenerate intima are not infrequently fatty as the result of the local toxæmia.

The functional and structural arrangements of the arterial walls thus necessarily entail some damage to more than one layer in all but the very slightest lesions. *In advanced lesions all the layers are abnormal*, though the degree to which each is involved may vary, and *this occurs irrespective of the site of the initial lesion*.

A clear distinction must be drawn between the two main groups of arterial disease. One is *diffuse* and affects the whole of the arterial tree. The other is *focal* or *nodular*, and affects only a part. The causes and the results of the two are totally different. The second group comprises atheroma, mesarteritis, endarteritis obliterans, and medial calcification. The first is arterio-sclerosis, using the term in its restricted sense.

Arterio-Sclerosis

Arterio-sclerosis is a diffuse affection, and all the arteries and capillaries, and, according to some writers, the veins as well, are abnormal. The lesions are best marked in the smaller vessels, whose walls are distinctly and uniformly thickened. The medium-sized arteries (renal, etc.) are also thickened, and their consistence may be firmer than normal. The aorta is thickened generally and, in addition, in old-standing cases, shows many atheromatous patches. In the early stages the resilience of the vessels may be good, but in advanced cases it is distinctly impaired.

Morbid Anatomy.—Microscopic examination shows alterations in all the three layers. The *intima* is usually more or less thickened (Fig. 261). There is always great hypertrophy of the elastic fibrils, and two or more laminæ, sometimes of considerable thickness, may be evident. In older cases de-

generative changes are present, the elastic tissue becoming granular and the connective tissue hyaline and sparsely nucleated, but the gross fatty and calcareous deposits which are met with in atheroma are rarely, if ever, seen. The *media* is always abnormal. In early cases it is thicker than usual, but the relative proportions of muscle, elastic and connective tissue are preserved, the only alteration being the hyper-



FIG. 261.—ARTERIO-SCLEROSIS: ELASTIC TISSUE STAINED TO SHOW THE HYPERPLASIA IN THE INTIMA. ($\times 75$.)

trophy (Figs. 262, *a* and *b*). In other cases it may be thinned, the muscle cells being atrophied and perhaps fatty; while in a third group the connective and elastic elements are in excess and the muscle cells are degenerate and few in number, in advanced cases only persisting along the edges of the coat (Fig. 263). They are most numerous and least altered along the outer margin, but may persist along the inner edge, while those in the centre have disappeared. The connective tissue

is usually hyaline or granular, and sparsely nucleated ; and the elastic fibrils, though excessive in number, are degenerate

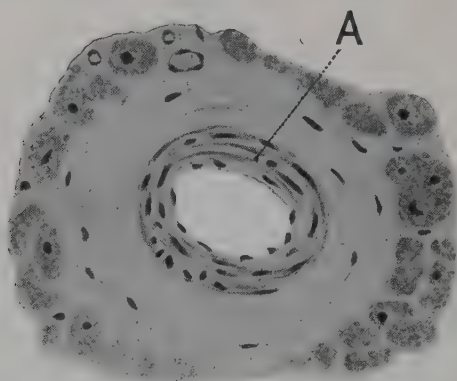


FIG. 262 (a).—ARTERY OF NORMAL HEART, SHOWING THE RELATIVE THICKNESS OF THE MUSCULAR COAT (A).

Contrast with Fig. 262 (b).

and granular (Fig. 264). The *adventitial* changes are less variable. The outer coat is almost invariably thickened,

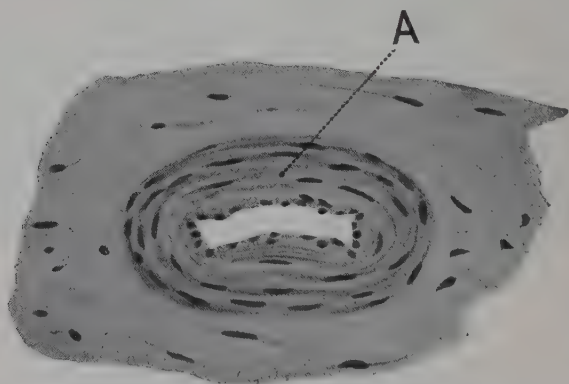


FIG. 262 (b).—ARTERY OF HEART FROM A CASE OF RENAL CIRRHOSIS IN WHICH THE HEART WEIGHED 18 OZ.

The media (A) is much hypertrophied.

being in the early stages thickly nucleated, and in old-standing cases hyaline and sparsely nucleated. The elastic tissue, here as in the other layers, is always excessive.

The adventitial thickening is the only constant change in these cases. If the media is thickened or fibroid, the intima may be but little abnormal; but if the former is atrophied, the latter is generally hypertrophied. William Russell states that the changes in the intrarenal vessels tend to intimal thickening, while the medial hypertrophy is best marked in the extrarenal vessels; but this may be due to the renal lesion

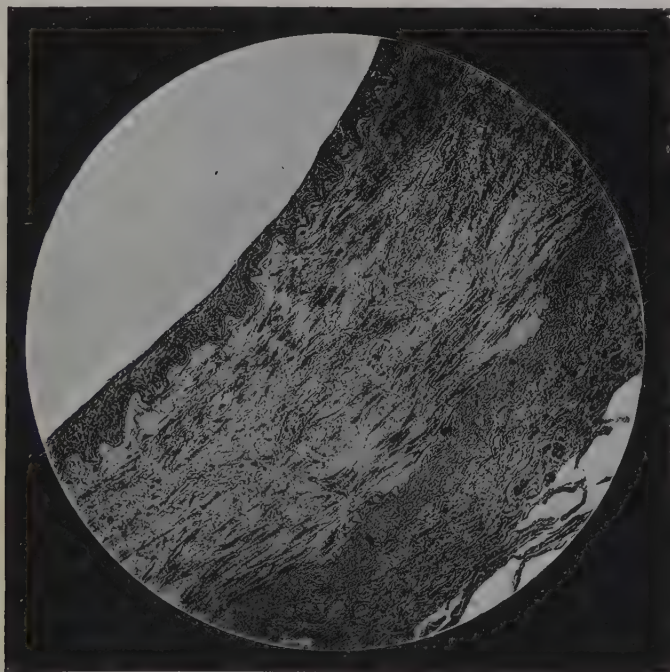


FIG. 263.—ARTERIO-SCLEROSIS.

The media is fibroid and only a few muscle fibres persist along the margins.
($\times 50$.)

being of older date. There has been considerable discussion in the past as to the precise histological details of the arterial lesions. But it is now generally agreed that medial hypertrophy (hypermyotrophy) is an early and essential phase, that it occurs throughout the whole arterial system, and that fibroid changes supervene both here and in the adventitia at a comparatively early period.

The capillary vessels are also abnormal. A healthy capillary cut transversely is apt to escape observation, the wall being a mere hair-line ; but in the cases which we are considering, a double contour is usually visible, the wall being several times its usual thickness ; the lumen may be somewhat narrowed.

The Etiology of Arterio-Sclerosis.—It has been known for long that the blood-pressure in cases of cirrhotic kidney

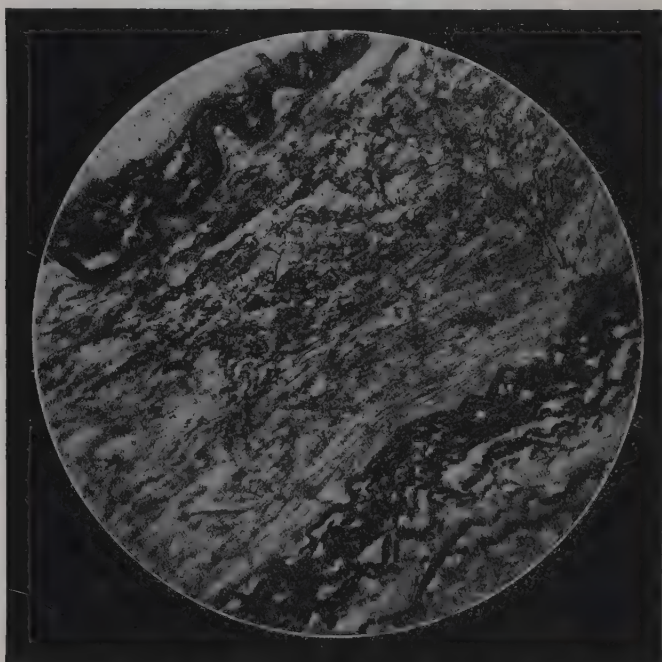


FIG. 264.—ARTERIO-SCLEROSIS. ($\times 75$.)

The same vessel as Fig. 263, stained to show the degenerate new-formed elastic fibres.

is almost always elevated, sometimes to an extraordinary degree, and it was at first supposed that this was due to the associated arterial disease. In 1903, however, Sir Clifford Allbutt¹ urged that arterial disease was not the cause of an elevated blood-pressure, but rather its result. "The pressure may be, and often is, low throughout the course of arterial

¹ *Lancet*, 1903, ii., 170, 329, 472, 645.

degeneration; or, again, having been high, it may fall; though, of course, in some elderly persons with arterial decay, occasional transient attacks of high pressure, such as occur at times in all of us, may be observed." Conversely, high blood-pressure cannot be maintained for long without arterial strain; and *arterio-sclerosis results from long persisting high blood-pressure of whatsoever origin.*

The process is well seen in cases of nephritis. In acute nephritis the blood-pressure is almost invariably elevated in the presence of symptoms, and falls with their remission, reaching normal readings in the majority of cases long before the albuminuria disappears (Fig. 266). In the chronic forms

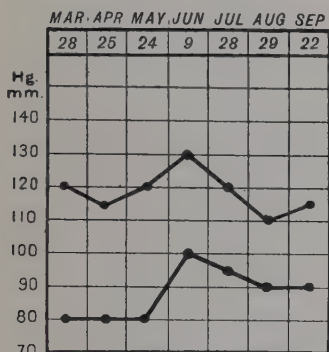


FIG. 265.—BLOOD-PRESSURE CHART.

Male, aged twenty-three. Large white kidneys weighing 16 oz. The heart weighed $6\frac{1}{2}$ oz.

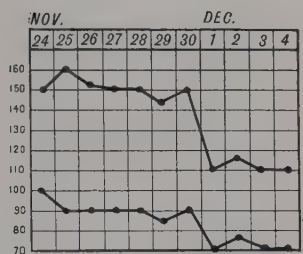


FIG. 266.—BLOOD-PRESSURE CHART.

Male, aged twenty-seven. Acute nephritis. Good result.

of nephritis the blood-pressure is not invariably elevated, and is, indeed, as a rule low in cases of large white kidney (Fig. 265), but it is always high in contracted kidneys (Figs. 267, 268), whether these are primary or follow an acute attack. In cirrhotic kidney, as Dickinson has shown, the whole of the arterial tree is affected, and the walls of the aorta and the great vessels, as well as the smaller branches, are notably thickened (Fig. 269).

The elevation of pressure in these cases is due to an increase in the peripheral resistance, which can only result from one of two causes: *a diminution in the calibre of the vessels, or an*

increased viscosity of the blood. Arterial disease does in many cases narrow the lumen, but it also sometimes widens it, and if it lessens it in some places, the local narrowing may be compensated by dilatation elsewhere. In syphilitic disease of the arteries, the lumen is often greatly narrowed, though the blood-pressure may never be above the normal.

The second factor, increased viscosity of the blood,¹ may

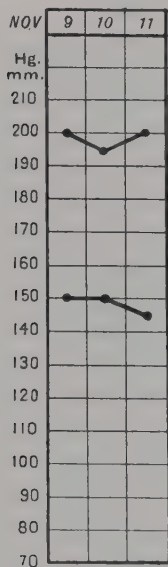


FIG. 267.—BLOOD-PRESSURE CHART.

Male, aged forty-three. Small white kidneys weighing $12\frac{1}{2}$ oz. The heart weighed 18 oz.

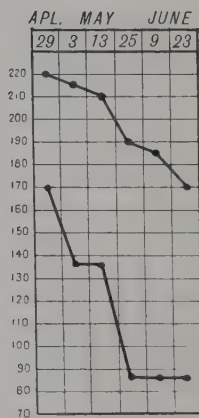


FIG. 268.—BLOOD-PRESSURE CHART.

Male, aged fifty. Small red kidneys weighing 8 oz. The heart weighed $21\frac{1}{2}$ oz.

conceivably be active in certain cases, but accurate observations on this point are difficult to obtain, for the methods of estimation are not above suspicion, even in laboratory experiments, and many diverse factors which may influence it, but cannot be accurately measured, come into operation in the wards. The viscosity of the whole blood rises steadily during the period of growth, and is higher in men than in women.

¹ Lyon, D. Murray, *Quart. Journ. of Med.*, 1921, xiv., 398.

It is affected by menstruation and pregnancy, by sweating exercise, by altitude, by variations in the red-cell and the white-cell counts, and by the percentage of oxygen and of carbonic acid in the blood, as well as by the saline and the colloid content; while it has no constant relation to the specific gravity of the blood or the height of the blood-pressure.¹

The viscosity is almost always increased in polycythæmia, and Parkes Weber² and others have shown that it may be twice or thrice, or even four times, greater than normal in cases of erythræmia, in which disease the blood-pressure is usually increased, apparently as a consequence; for, although this might be due to renal disease, which is a common accom-



FIG. 269.—DIAGRAM, DRAWN TO SCALE AND REDUCED, SHOWING THE SIZE OF THE ARTERIES AND THE THICKNESS OF THEIR WALLS IN A SERIES OF NORMAL PERSONS AND CASES OF GRANULAR KIDNEY (W. G. DICKINSON, *Occasional Papers*, 1896, 196).

paniment in these cases, the kidneys may be normal, as in Case 21 of Weber's series, in which the heart was "somewhat hypertrophied."

In one group, the polycythæmia hypertonica of Geisbock and Hess, the same relationship apparently obtains. We have seen one case of this kind.

The patient, a solicitor aged thirty-nine, had been steadily losing strength and flesh for at least a year, and had been noticed to be "blue" in colour for the same time. He had had several attacks of "influenza," in which he was fevered and frequently shivered, and experienced very severe headache, while there was but little catarrh;

¹ Allbutt, Sir Clifford, *ibid.*, 1910-11, iv., 342; Welsh, W. H., *Heart*, 1911-12, iii., 118.

² Parkes Weber, *Quart. Journ. of Med.*, 1908-9, ii., 85.

and during these illnesses he became notably cyanosed, while his urine contained much albumin. He was a well-built but badly nourished man, and sweated profusely. The cyanosis was always considerable, and once, during cold weather, was extraordinary in its degree, and, although general, much more intense in the head and neck than elsewhere. The jugular veins were turgid and prominent. The heart was slightly enlarged, the second aortic sound loud and ringing, and the peripheral arteries were thick and tortuous. The retinal arteries were of characteristic silver-wire appearance. The systolic blood-pressure measured 175-180 mm. Hg. The liver and spleen were slightly enlarged, and the urine habitually contained albumin in varying amount, sometimes, however, only a trace. The red cells numbered 10,000,000, the hæmoglobin value was 120 per cent., and the white cells numbered 22,400 per c.mm. The average size of red cell was rather above the normal, and a few cells were certainly megalocytes. No erythroblasts were seen. The white cells were chiefly polymorphonuclear.

The patient himself was definite that his illness had lasted for some twenty years, though it was only recently that he had been seriously inconvenienced; and he had conducted a considerable business during this period. He was a married man with a healthy family. He had always been liable to bilious headaches, even as a boy at school, and these had gradually become more severe and of longer duration. They recurred every few weeks, and his tongue became coated and his bowels confined during their persistence. He was, he said, a "fair" boy, but his complexion darkened in the twenties, and in the early thirties he became "plethoric." His father, too, had been "blue" for the last twenty years of his life; but he had carried on business until shortly before his death at the age of seventy, after an illness of ten days' duration.

This patient died about two years later, and there was no *post-mortem* examination, so that the condition of his kidneys was not ascertained. But it seems probable that the polycythæmia was the primary event and the cause of the increased blood-pressure and the cardio-vascular hypertrophy.

There has been some difference of opinion as to the *site* of the increased peripheral resistance. Sir Clifford Allbutt thinks that it is in the capillary network as in normal individuals, while Sir Douglas Powell, Russell, and others maintain that it is situated in the finer arterioles. Broadbent and Sansom thought that both factors might be active. The last opinion seems probably correct, for although the first theory seems applicable to the polycythæmic group, vasodilator drugs can often in other cases relieve high pressure, at any rate temporarily, and their action can only influence the muscular arterioles.

The blood-pressure in elderly persons may be elevated without any evidence of renal lesions (hyperpiesis), and Sir Clifford Allbutt thinks that the most important cause is "repletion, relative or absolute—i.e., tempered or untempered by air and exercise." Although at first the elevation may disappear as the result of suitable treatment, it tends as a rule to recur and to become permanent, and ultimately to be associated with arterial thickening. William Russell has also brought forward evidence in support of the same theory. He differs from Sir Clifford Allbutt in his contention that the initial error is always an excessive ingestion of food greater than the work done requires or elimination can overtake, and suggests that in many cases the chief cause is an intoxication due to gastro-intestinal indigestion and the production of hypertonic substances. There seems to be little doubt that both observers have right on their side, but it seems certain that other causes may also be active, for the association of gout and chronic lead-poisoning with arterio-sclerosis has long been recognized, though the precise mechanism by which the blood-pressure is elevated is still undetermined. Renal disease, in these cases, though commonly present, may be absent, though the arterial lesions are well marked.

The isolation of adrenalin and the discovery by Josué of its action in producing arterial disease in rabbits have focussed the attention of many observers upon the suprarenals, and it has been demonstrated that these are very often enlarged both in chronic renal disease and in cases of arterial disease. The chief alterations have, however, been situated in the cortical parts of these organs, while it is known that adrenalin is the product of the medulla. But Josué has shown that substances obtained from the cortex, though not adrenalin, have also a hypertonic action upon the vessels, and maintains that cortical hyperplasia, however it may act, whether by some reaction preparatory to the secretion of adrenalin by the medulla, or by its own effects, is an index to the activity of the gland as a whole.

Thayer¹ has pointed out that the blood-pressure is often elevated following an attack of *enteric fever*, and states that

¹ Thayer, W. S., *Amer. Journ. Med. Sci.*, 1904, cxxvii., 391; *Johns Hopkins Hosp. Bull.*, 1904, xv., 323.

the average systolic pressure in such individuals is appreciably higher than that of healthy individuals of the same age, and that the radial arteries are palpable nearly three times as often as they are in healthy persons who have never had the disease. It is difficult to understand why this should occur, as the blood-pressure during an attack is low, and tends to fall rather than to rise. The small arteries, however, are not infrequently affected in the acute infections (*vide* p. 491), and the lesions are often associated with microscopic damage to the viscera (submiliary nodules in the heart, focal necrosis in the liver, cellular collections in the kidneys, etc.), and although the separate lesions are individually negligible, their collective result in cases where they are widespread may be considerable and may lead to such an increase of the peripheral resistance that an elevation of the blood-pressure is produced. The importance of infective diseases, and particularly of chronic septic infection of the rheumatic type, as a cause of arteriosclerosis, has been emphasized by Ophüls,¹ according to whom the arterial lesions attain their full development long after the active infective process has subsided.

Hypertrophy of the media, and probably of the adventitia as well, ensues as the natural sequel of the increased work imposed upon the vessels, but there is only a narrow margin between the degree which induces hypertrophy and that which initiates degenerative changes; and "sooner or later the body meets with circumstances in which either the nutrition is not maintained, or the stimuli acting on the media become greater than can be sustained" (Klotz), and degenerative changes ensue with atrophy of the "noble" muscular cells, and hypertrophy of the less highly specialized connective tissue, according to the usual rules. The chronic intoxication which is always present in these cases will, too, tend to produce degenerative lesions in the muscle cells. The causes of the intimal thickening, when it occurs, may be similar, or it may be due to continued irritation by toxic materials in the blood-stream.

THE FOCAL LESIONS

(1) **Atheroma** (*Endarteritis nodosa vel deformans*).—Atheromatous patches are almost invariably present in the arteries

¹ Ophüls, W., *Stanford Univ. Publ., Med. Sci.*, 1921, i., 1.

of elderly persons, though, of course, their number, size, and distribution vary in different cases. The patches are elevated above the surface with more or less abrupt edges, and even in small vessels rarely involve the whole circumference. Sometimes they are mere pin-points, sometimes plaques as large as a shilling, and they may, by coalescence, involve comparatively large areas. In the early stages they are soft and gelatinous and translucent; in the later stages opaque and

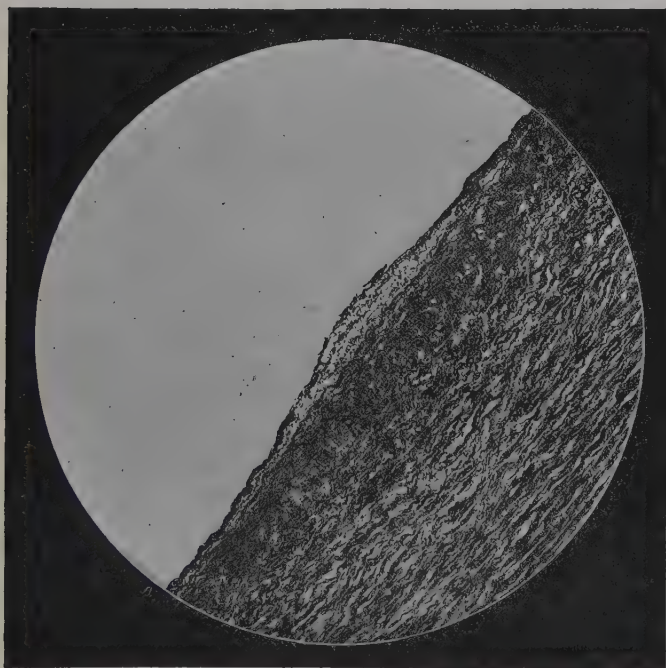


FIG. 270.—EARLY ATHEROMA.

The intima alone is thickened. Elastic stain. ($\times 40$.)

firm, or hardened by calcareous deposits. Ulceration is not uncommon, and thrombi may be deposited upon them.

It is generally agreed that the first portions of the aorta are most often involved, but there is a difference of opinion as to the frequency with which the other arteries are affected. The coronary arteries of the heart, the cerebral and the peripheral arteries, are, however, affected more often than those

of the abdominal viscera, and the pulmonary arteries are but seldom damaged, save in cases of chronic pulmonary or cardiac valvular disease.

The early lesions are found in the deeper layers of the intima (Fig. 270), but in advanced examples the whole thickness of the wall is found, on microscopic examination, to be involved, though the degree to which each layer is affected varies in different cases. The intima is always thickened, often greatly, and in the early stages is very cellular, the elastic



FIG. 271.—SYPHILITIC AORTITIS.

Man aged forty-nine. The orifice of the left coronary artery is almost occluded.

tissue sharing in the hyperplasia. In the later stages degeneration occurs, and fatty, granular, mucoid, hyaline, or calcareous changes may be found, always most extreme near, though not necessarily next, to the outer margin of the coat. The media is atrophied often to a notable degree, and may be entirely destroyed, and its elastic tissue may be excessive and degenerate. The adventitia as a rule is less abnormal than

the other coats, but it is sometimes grossly affected. In early cases it is thickened and very cellular, with little foci of mononuclear cells scattered here and there. In older examples it is distinctly sclerosed, and the new-formed tissue is more or less degenerate. Here, too, as in the intima and the media, elastic hyperplasia and degeneration may be distinct.



FIG. 272.—SYPHILITIC MESAORTITIS. ($\times 150$.)

The vasa in the adventitia show endarteritis obliterans, and are surrounded by many mononuclear cells.

The vasa vasorum often show gross disease, the arteries being affected by an endarteritis obliterans, and the veins may show thickening of their coats.

(2) **Patchy Mesarteritis.**¹—The naked-eye appearance of this form resembles in some degree the atheromatous lesions. The patches in the early stage are elevated and soft, and have

¹ The description of these lesions is based upon the syphilitic form.

a greyish gelatinous appearance, and are generally most numerous in the first part of the aorta (Fig. 271). In older cases they are tough and fibrous and pearly white, and may present a depressed, stellate-shaped scar (Figs. 29 and 303). Calcareous deposits are infrequent, and are never massive, and ulceration does not occur.

The microscopic picture is characteristic. In the early

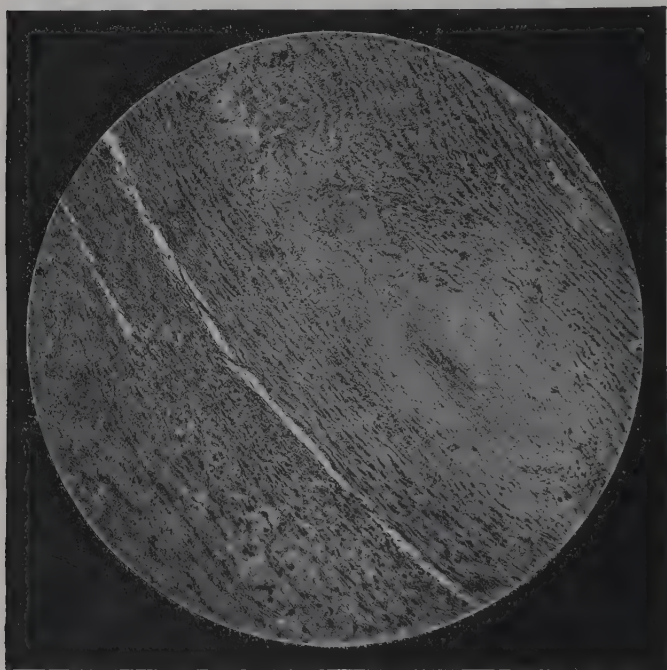


FIG. 273.—SYPHILITIC MESAORTITIS, SHOWING A PATCH OF NECROSIS IN THE MEDIA. ($\times 90$.)

stage the vasa in the adventitia are surrounded by clumps of mononuclear cells, and their walls are infiltrated and thickened, and their lumen is narrowed (Fig. 272). The media shows areas of necrosis (Figs. 273, 274) into which new-formed branches of the vasa penetrate, surrounded by a very cellular granulation tissue; and the intima is thickened and cellular opposite the patch. In older specimens the adventitia is thickened and sclerosed, and the vasa may be almost com-

pletely obstructed; the media may have disappeared completely in places and be replaced by new-formed connective tissue (Fig. 275), and the intima, invaded by the vasal branches, though thickened to a greater or a less degree, may show some cicatricial shrinking.



FIG. 274.—SYPHILITIC MESAORTITIS (ELASTIC STAIN), SHOWING THE DEGENERATION OF THE ELASTIC TISSUE IN A GRANULOMATOUS PATCH. ($\times 90$.)

(3) **Endarteritis Obliterans.**—Endarteritis obliterans occurs chiefly in the smallest arteries and the arterioles; it is often present in the cerebral arteries and in the vasa of the aorta. It is always local in its distribution, and never involves more than a short length of a vessel, but as the whole circumference of the wall is involved, the lumen is always narrowed and sometimes completely blocked. Its clinical importance is, in consequence, considerable.

The intima is always greatly thickened, the new-formed connective tissue being in the early stage loose and very cellular, with much hypertrophy of the elastic elements (Fig.

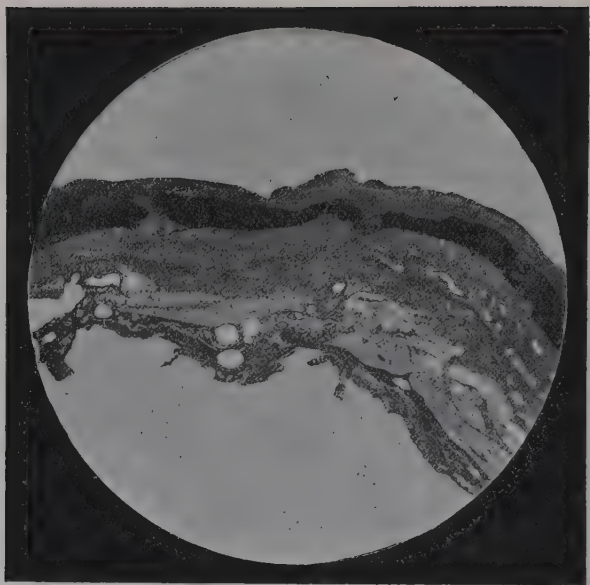


FIG. 275.—SYPHILITIC MESAORTITIS (ELASTIC STAIN). ($\times 8$.)
The media (the thick black line) is completely destroyed in two places.

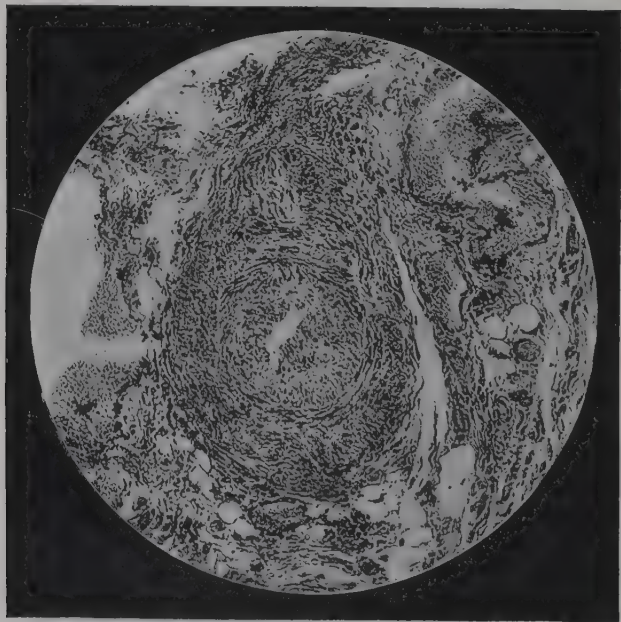


FIG. 276.—ENDARTERITIS OBLITERANS. ($\times 150$.)
(From a case of acute rheumatism.)

276), but in the later stages poorly nucleated and hyaline, with degeneration of the new-formed elastic fibres. The media may be but little altered or definitely atrophied. The adventitia is always thickened, sometimes to an extraordinary degree (Fig. 277), and, like the intima, shows new formation

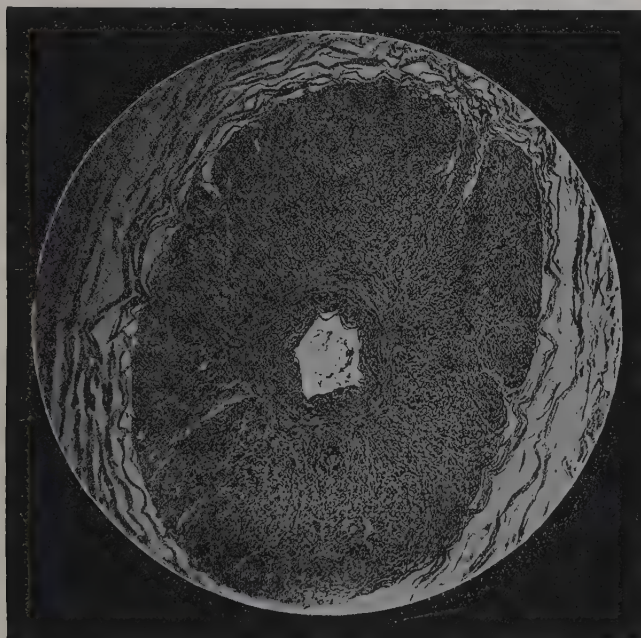


FIG. 277.—SYPHILITIC ENDARTERITIS OBLITERANS.

The adventitia is greatly hypertrophied. ($\times 90$.) (A. W. H.)

of elastic tissue. The relative thickness of the intima and the adventitia varies considerably in different cases, and sometimes one, sometimes the other, shows the greater change. Gross fatty or calcareous degeneration is rarely met with.

(4) **Medial Calcification.**—Medial calcification occurs in two forms. In those cases of atheroma in which extensive calcification of the thickened intima has occurred, the medial layers in the immediate vicinity may be similarly affected. It also occurs, however, in a widespread manner, though still patchy in distribution, without any, or with only minimal,

alterations in the intima. In this case it is most often found in the peripheral arteries, and is readily detected during life by the finger, which, as it runs along the vessel, meets now a hard and now a soft patch (Figs. 278, 279). The vessels are frequently dilated. On microscopic examination in well-



FIG. 278.—MEDIAL CALCIFICATION. (J. R. R.)
(Skiagram showing the ulnar and radial
arteries.)

marked cases the proper structure of the media has wholly disappeared, and is replaced by a mass of calcareous granules, often welded together and encircling the vessel. In the larger vessels the whole of the circumference is rarely affected.

The Etiology of Focal Lesions.—

Local lesions own a local cause, but it must be admitted that our information as to the precise etiology of focal arterial disease is at present imperfect. The distinction between athetoma and patchy mesarteritis is important. In the former the primary lesion is intimal hyperplasia,

and the media atrophies before the pressure of the nodule under the influence of the intra-arterial blood-pressure. In the latter the primary lesion is in the middle coat. Patchy mesarteritis is most frequently due to syphilis, and represents the reaction produced by the invasion of spirochætes (which have been demonstrated *in situ* by Schmorl, Burda,

Wright, and Klotz), or follows obstruction of the vasa in the adventitia and resultant medial necrosis. But while early lesions may be accurately classified, *the ultimate scars are essentially similar*, and the question must be approached in a general way.

Evidence is steadily accumulating in favour of the view that the infections as a whole are a not uncommon cause of local lesions. It has been known for long that in cases of pyæmia patches similar in their nature to those sometimes found on the valves of the heart may be present on the arterial walls, and that an infective arteritis may occur by extension from a neighbouring infective focus (Fig. 175). As a rule, however, bacteria are not found in early specimens of atheroma, and spirochætes, too, are usually absent in cases of syphilitic disease, though tubercle bacilli, pneumococci, anthrax bacilli, streptococci, and staphylococci have all been found in exceptional cases. Usually the infection takes place from the blood-stream, but an embolic origin is conceivable, and an acute mesarteritis has been observed in acute rheumatism, enteric fever, and diphtheria. Plaques of recent atheroma are often present in the aortas of individuals who have died from acute disease. They have been reported in cases of smallpox, diphtheria, erysipelas, pneumonia, acute rheumatism, influenza, scarlatina, measles, and enteric fever, being present in the latter in twenty-one of fifty-two *post-mortems* (Thayer and Brush). Experimental research, too, has shown similar results, and intimal changes have been produced by inoculations with streptococci, medial changes with diphtheria toxin and the *Micrococcus rheumaticus*, and intimal and medial changes in conjunction with staphylococci.

Josué's experiments with adrenalin opened out a new field

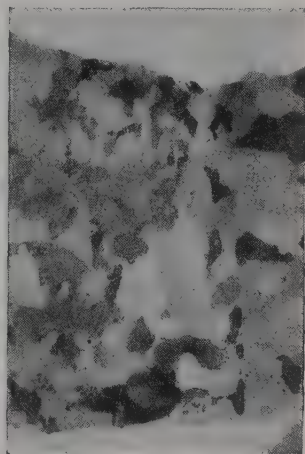


FIG. 279.—RADIOGRAM OF AORTA, SHOWING CALCAREOUS DEPOSITS. (K. M. C.)

of observation. He showed that repeated injections of this drug into rabbits produced local thinning and calcification of the aortic media with, in some cases, a local thickening of the overlying intima; and his results have been confirmed by numerous observers. Other drugs, too, of similar hypertonic action (nicotine, digitalin, hydrastine, barium chloride) have also been shown to produce similar results, and Croftan has produced various lesions in the vessels with solutions of xanthin and hypoxanthin.

The exact mechanism of these procedures is not yet understood, but Klotz has shown that medial calcification may follow an increase of the blood-pressure produced by suspending rabbits by the hind-legs for short periods over many days, the lesions being confined to those vessels (carotids and aorta) in which the blood-pressure was raised, and absent in those below the diaphragm; atheromatous lesions, too, were found in the carotid arteries, which suggests that both medial and intimal lesions may result from elevation of the blood-pressure.

Although it is not difficult to demonstrate the various ways in which focal lesions may be produced, it is not easy to define their actual causes, taking into consideration their frequency in persons over middle age and their special sites of election in the arterial tree.

The frequency with which focal lesions occur makes it evident that some of the possible causes which we have considered have but little to do with their origin. Tuberculosis and syphilis, for example, must be ruled out of court, for these diseases, though prevalent enough, are by no means universal. The infections as a whole can hardly be responsible for many cases. It is true that acute arterial lesions are not infrequently met with in these diseases, but *post-mortem* records show that gross changes are, on the whole, uncommon, and that when present they are rarely extensive. The frequency, however, with which infections occur during life, the exanthemata in childhood, and influenza, pneumonia, enteric fever, etc., in later years, make it possible that their influence is greater than has been otherwise supposed, the little lesions thus initiated allowing, as places of lessened resistance, other

causes to produce greater results than would otherwise be possible.

Nodular lesions are not uncommon in cases of diffuse arteriosclerosis. The lumen of the smaller vessels is often narrowed, and the fibrous overgrowth of the adventitia sometimes spreads into the tissues and produces little islets of fibrosis in the viscera, entangling and destroying the adjacent cells. Any of the organs may be affected, but the heart and the kidneys are chiefly involved. It seems probable that in arteriosclerosis nodular lesions may arise in the larger vessels, such as the aorta, from a similar affection of their vasa.

On the whole, however, the explanations seem insufficient, and other causes must come into action.

The special distribution of the focal lesions has long been held to indicate that mechanical irritation plays an important part in their etiology. The buttressing of particular parts of the arterial system shows that special stress obtains at special points even in normal circumstances, and certain vessels, such as the coronary arteries of the heart, the intestinal arteries and those of the limbs, must be more liable to injury than those contained within organs which move but little or are deeply situated. Popliteal aneurysms, for instance, seem almost to have disappeared with the post-boys, and tortuosity of the temporal arteries is of common occurrence, as it is produced by the pressure of the hat against the skull. It is true that provision seems to have been made for such varying needs, but the continued slight trauma of the systolic pulse waves must nevertheless exert an influence which in time will produce damage, and this is most likely to occur in cases where physical labour is the chief occupation. Medial calcification is thus common in the femoral and iliac arteries of persons such as policemen, who are constantly standing, and in the brachial and radial arteries of individuals whose work entails continued use of their arms, and is even, according to Klotz, more common on the right side in right-handed persons. Des Ligneris has reported cases of intimal thickening in the femoral and external iliac arteries which he considers are due to mechanical causes, the pressure of the blood-stream against the posterior wall when the hip is flexed, or the mechanical stretching when the legs are moved. The arterial blood-

pressure is liable to many alterations from physiological influences, and these are almost always in the direction of temporary elevation above the normal. Exercise, the ingestion of food, cerebral excitement, etc., all produce an increase, and this may be considerable if the stimulus is great or frequently repeated, and the probability of injurious irritation will, under these circumstances, be greater than in normal conditions.

The form and site of the initial lesion depend upon several factors. In some instances overuse of limbs leads to their special involvement; in other cases the aortic strain may be greatest; in others, again, a previous lesion may determine a particular site.

The special liability of the aorta and the cerebral arteries to syphilitic disease has not yet obtained a satisfactory explanation, but it may be pointed out that their structure is very specialized, and that the aorta is exposed to the full force of the ventricular systole, while disease of the cerebral arteries is so prejudicial to the proper working of the brain, and even to life itself, that it has probably attracted more than its fair share of attention.

Endarteritis obliterans is not always of pathological significance. It occurs, for instance, in the umbilical vessels after their special function has ceased; in the vessels of a stump after amputation of a limb; and in arteries whose lumen has been narrowed or obstructed by any cause, the normal proliferative changes exceeding the degree which is required.

It is of common occurrence in the infections, and has been found in acute rheumatism, diphtheria, scarlatina, smallpox, and enteric fever. It is particularly frequent in tuberculosis of the cerebral membranes and in syphilis, the adventitial changes in the latter case being usually extreme.

Medial calcification is most frequently associated with mechanical sources of irritation.

CHAPTER XXIX

THE SYMPTOMS OF ARTERIAL DISEASE

THE symptoms of arterial disease vary greatly in different patients, as they depend both upon the variety of the arterial lesion and upon its special site, and are, in the majority of cases, the results of defective function of the various organs rather than an indication of the process which has produced them. The symptoms of an aneurysm, for example, arise chiefly from pressure upon neighbouring structures, and tumour and pulsation are the only evidence of the arterial dilatation.

The two main groups of arterial disease contrast in almost every particular. In the *focal or nodular group* the lesions are always local and never of great size, although they may be numerous, and by coalescence affect considerable tracts of the vessels. The larger arteries are chiefly involved. In atheroma and mesarteritis the wall is weakened, and dilatation of the vessel, or even rupture, may ensue. In medial calcification the artery is often wider than normal. In endarteritis obliterans the arterial lumen is narrowed, and thrombosis often follows, a result which is unusual in the others.

In *arterio-sclerosis*, on the other hand, the whole of the arterial tree is involved, and the change is most considerable in the smaller vessels. The walls are thickened and on the whole strengthened, so that dilatation or rupture is uncommon save in the cerebral arteries, where miliary aneurysms, the common cause of cerebral hæmorrhage, are often found. Thrombosis is comparatively rare, but the lumen is generally narrowed, and the visceral blood-supply may be limited in consequence.

The focal lesions may be widespread, and even extreme, without causing any abnormal strain upon the left ventricle. In other cases, however, where the thoracic aorta or the

splanchnic arteries are involved, the left ventricle may be considerably hypertrophied. In *arterio-sclerosis*, on the other hand, the left ventricle is always hypertrophied, sometimes to an extraordinary degree; and, as in valvular disease, the reserve power is necessarily more limited than in health, and the "field of response" is correspondingly diminished. The essential cause of arterio-sclerosis is continued elevation of the arterial pressure, and the arteries as well as the heart are at first thickened by muscular hypertrophy. In time, however, fibrosis supervenes, perhaps as a result of the toxæmia, and their elasticity is largely lost.

One must distinguish between the two types of arteries: One set are the "mains" for the distribution of the blood, affording an elastic assistance to the heart, and insuring a continuous flow, at equable pressures, through the capillaries. The other set are the "supply-pipes," with "taps" which can be shut, at any rate partially, and thus alter the distribution of the blood among the various organs as necessity dictates. If the mains are rigid, the heart has more work to do, and the capillary current is less even in its flow. If the "supply-pipes" are stiff, the "taps" cannot be altered at discretion, and variations in the functional demands of the tissues can only be inadequately supplied, and that by variations in the general blood-pressure and an increased strain upon an already over-taxed ventricle.

The symptoms in the two groups arise in different ways. In the *focal* cases they are the results of *hæmorrhage*, *thrombosis*, or *arterial dilatation*. The results of hæmorrhage vary mainly with its amount, and also, to some extent, with its site, cerebral hæmorrhage, for instance, causing death with a loss of blood which, if external, would occasion little, if any, discomfort. The results of thrombosis depend both upon its site and upon the rapidity of the closure. Sudden occlusion of a coronary artery may produce myocardial infarct or rupture of the heart, while a gradual narrowing may allow of compensatory dilatation of anastomotic vessels, with but little interference with the nutrition of the cells involved. The results of dilatation depend mainly upon its extent and its site, and are most marked when it obtains within a cavity of fixed dimensions—*e.g.*, within the cranium or the thorax.

The lesions which are thus produced are as a rule isolated and single, though sometimes of considerable size.

In *arterio-sclerosis* visceral lesions are widespread, multiple, and individually of trifling degree, though their collective influence may be great. Dilatation and hæmorrhage are unusual, save in the case of the cerebral arteries, and thrombosis is also infrequent. But the lumen of the vessels is often narrowed, and the visceral blood-supply is in consequence diminished—an effect which is intensified by the limitation of the response to vasomotor influences entailed by the fibrosis of the vessel walls. This fibrosis, too, not infrequently spreads into the tissues from the adventitia, and produces little islets of fibrosis in the organs, entangling and destroying the adjacent cells. Any of the viscera may be involved, but the heart and the kidneys are the common sufferers. Renal changes are the rule in arterio-sclerosis. The initial fault is often renal, a cirrhosis of acute or chronic type; and a somewhat similar condition, the arterio-sclerotic kidney, is generally more or less evident in cases which have arisen from other than renal fault.

The symptoms of the two groups contrast correspondingly. In the *focal* group aneurysm, infarct, and hæmorrhage are the essential conditions. In *arterio-sclerosis* the heart is always overtaxed, renal excretion is generally inadequate, and other viscera are apt to be damaged; while their function is still further impaired by the limitation of their blood-supply produced by the arterial disease and the relative cardiac insufficiency. At first symptoms only occur on special strain, whether physical, mental, or visceral; but ultimately even the normal work is imperfectly performed. The onset of symptoms is thus insidious, unless some special cause precipitates their advent. In the focal group symptoms often ensue suddenly, and are acute from the outset.

From yet another standpoint the two groups contrast. The causes of the first group are numerous—syphilis, tuberculosis, trauma, intoxications, and infections of various kinds; and while some of these may be continuous in their action, others have only a limited period of harmful activity. Focal lesions may thus be progressive, or merely the healed scars of ancient wounds. *Arterio-sclerosis*, on the other hand, is due to con-

tinued high arterial pressure ; and although in some cases the initial elevation is probably of functional origin, and therefore removable on removal of its cause, in others it is secondary to visceral lesions which are incurable. When once permanent changes have occurred in the vessels, the high pressure is permanent ; the strain upon the heart and vessels continues ; and, as time goes on, the lesions inevitably progress.

The symptoms in arterial disease may thus be produced in various ways, and as they may be related to almost any organ, and may simulate even closely symptoms due to other pathological lesions, their exact significance may be difficult to determine. This is particularly the case with the focal diseases. In arterio-sclerosis the underlying general condition can in most cases be readily recognized.

The essential and constant feature in arterio-sclerosis, at any rate until cardiac failure is well marked, is *elevation of the aortic blood-pressure*. In a large group of cases, renal cirrhosis occurs, if not antecedent to, at least *pari passu* with, the cardio-vascular changes which are compensatory and permanent. Sometimes the cardio-vascular hypertrophy is not well marked, and death then seems to ensue more rapidly than is the rule when the hypertrophy is considerable. The cause of the failure may lie in anæmia and debility consequent on the renal disease or other coincident causes, but is sometimes, at any rate, congenital in origin. We cannot all of us become Sandows, even if we follow a similar line of training, and the vascular musculature is in many ways comparable to the muscle of the limbs.

The introduction of the Riva-Rocci sphygmomanometer and its numerous modifications has put within the reach of the practitioner a method by which the arterial pressure may be measured with fair accuracy, and without any special difficulty of technique. But while it is useful to have figures which can be contrasted, and so give a measure in some way of the severity of the disturbance, it must be remembered that the blood-pressure varies to a considerable extent in the healthy individual from causes of a temporary and often trivial nature (emotion, exercise, diet, etc.), the influence of which cannot always be accurately correlated, and that similar results may

be noted even in well-marked examples of high blood-pressure. The earlier readings in hospital patients confined to bed are often shown by continued observation to be misleading, from causes of this temporary kind, and but little importance should be attached to isolated observations (e.g., those of Life Assurance reports) though daily records are of considerable value.

The information which is desired can, too, be obtained from other data. Digital examination of the pulse is at the present day too much deprecated, and while we must acknowledge that our impressions of the arterial pressure from such examinations have not infrequently been at variance with instrumental readings, a constant comparison has led to greater accuracy. The error seems to lie in the failure to appreciate that the size of the pulse wave and the amount of the pulse pressure¹ have no constant relation to the height of the aortic systolic pressure. To take a concrete example the large quick pulse of aortic incompetence may be of much lower pressure than a small but wiry pulse (Figs. 280, 281).

The sphygmomanometer, too, does not give the information with regard to the condition of the arterial walls that is readily afforded by the finger, which can detect local as well as general thickenings. If all the accessible arteries (retinal, radial, brachial, temporal, facial, femoral, dorsalis pedis) are explored, a fairly accurate idea of the condition of the vessels as a whole may be obtained. It is true that the condition of the peripheral and the visceral vessels by no means always corresponds, and changes may be extreme in one set without the other being seriously involved, but on the whole the comparison is fairly accurate. The failure in correspondence is seen chiefly in the focal lesions; in arterio-sclerosis the association is more absolute, so that, while irregularities in the vessel walls convey only a limited significance, general thickening is always important. The ophthalmoscopic evidence may be unmistakable.

Examination of the heart is, of course, equally essential. A well-sustained apex impulse displaced downwards into the sixth or seventh interspace, prolongation and dulling of the first sound, and accentuation of the second aortic sound are, in the absence of valvular flaws, positive evidence of high aortic

¹ The pulse pressure is the difference in pressure between the systolic and the diastolic pressures.

blood-pressure. The second aortic sound may be intoned and ringing in quality, but this is probably due to aortic dilatation, whether local or diffuse.

It is easy to recognize these cases when they come under

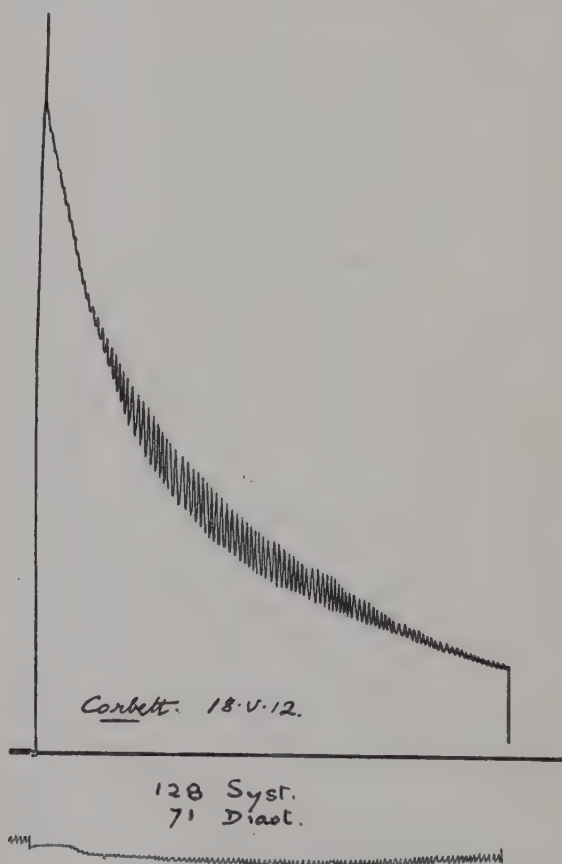


FIG. 280.—BLOOD-PRESSURE TRACING (GIBSON SPHYGMOMANOMETER):
AORTIC REGURGITATION.

The pulse is much larger than in Fig. 281, though the systolic blood-pressure is much lower. The pulse pressures are equal.

observation from casual reasons, such as insurance examination, etc., at a time when the compensatory mechanism is well established and sufficient, and symptoms are in abeyance, but an accurate diagnosis may be extremely difficult in the

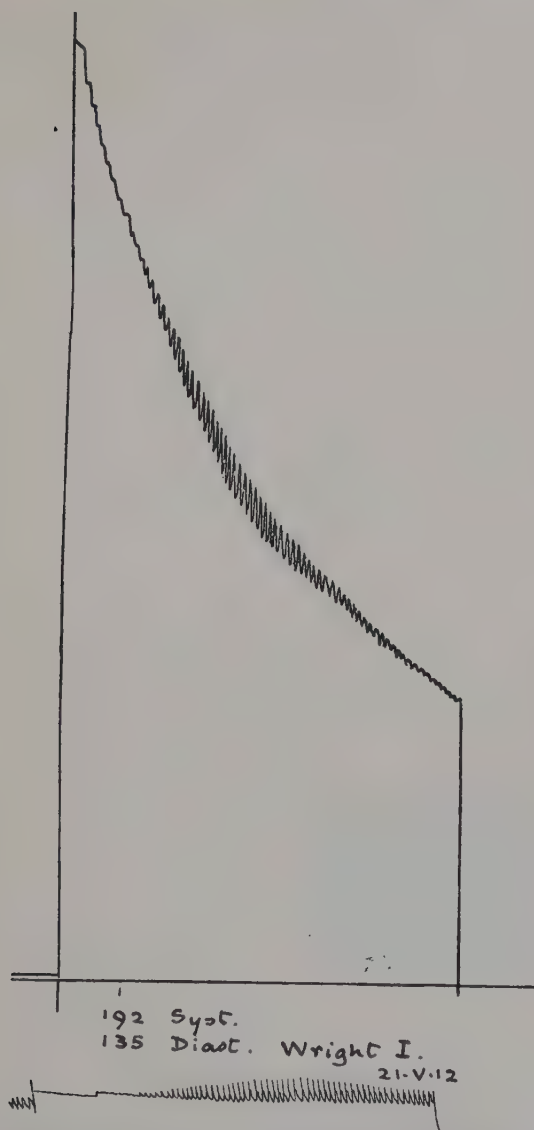


FIG. 281.—BLOOD-PRESSURE TRACING (GIBSON SPHYGMOMANOMETER):
CHRONIC NEPHRITIS.
Contrast with Fig. 280.

later stages if cardiac failure has supervened. The blood-pressure may be within normal limits, the second aortic sound

may not be notably emphatic, and the first sound may be short and sharp, or obscured by murmur if the mitral valve has given way before the strain.

Cardiac hypertrophy is not necessarily associated with high arterial pressure. In well-compensated cases of mitral incompetence and aortic stenosis, for example, the degree of ventricular hypertrophy is that required to ensure the entrance into the aorta, in the proper time, of the proper amount of blood at the proper pressure. In aortic incompetence other factors come into operation. If an average amount of blood

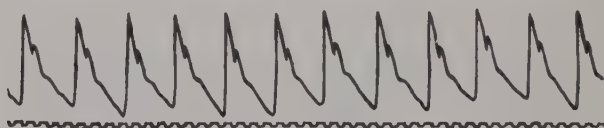


FIG. 282.—SPHYGMOGRAPHIC TRACING.

Aortic incompetence.

is to be left in the arteries at the end of diastole, the amount which is thrown into them during systole must be greater than normal, so as to allow for that which returns into the ventricle as soon as systole has ceased; and the rapid fall of pressure during diastole requires an excessive systolic pressure.

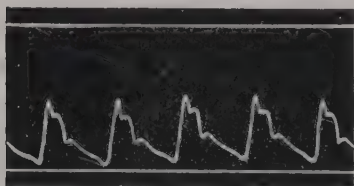


FIG. 283.—SPHYGMOGRAPHIC TRACING.

Normal.

The pulse pressure is the true index of the degree of the mechanical fault. But such a pulse differs notably from that associated with granular kidneys. In aortic incompetence, as sphygmographic tracings show so well (Fig. 282), the pressure falls rapidly, and the mean pressure is low relatively

to the maximum.

A high blood-pressure is not invariably evidence of cardiac sufficiency, and may, indeed, be accompanied by definite signs of cardiac failure. The most striking example which we have observed occurred in a man, aged fifty-one, who was complaining of breathlessness and slight œdema of the feet. The sudden occurrence of a left facial palsy, accompanied by

some difficulty in speech, followed a few days later by a partial right hemiplegia, was considered to be the result of cerebral hæmorrhage, as the systolic brachial pressure varied from 170 to 210 mm. Hg. But *post-mortem* examination revealed in the cortical vessels multiple emboli which were derived from clots in the dilated left ventricle. The highest pressure which we have observed—270 mm. Hg.—occurred in a man who became breathless on the least exertion, and developed œdema in the feet and legs whenever he was allowed out of bed. In another patient, who was admitted to hospital suffering from hemiplegia, the symptoms were due to cerebral softening from thrombosis, though the systolic blood-pressure was 245 mm. Hg.

Such failure is, of course, relative and not absolute. The ventricular wall may be greatly thickened, and the cells may be normal on microscopic examination, but the heart may yet be overtaxed and unable to accomplish its necessary work.

Cardiac strain may arise in two different ways. The heart may be unable to overtake extra work imposed upon it, or may from intrinsic causes (fibroid, fatty degeneration, etc.) be unable to accomplish even a normal amount. The arterial pressure may thus vary either upwards or downwards in the presence of symptoms, and it may be impossible for a time to say whether the actual pressure is too high or too low for the particular individual. If improvement occurs, the pressure may rise or fall; as death approaches, it may also rise or fall. In Fig. 284 the pressure is seen to fall coincidentally with improvement in the general condition; in Fig. 285 the pressure rose with improvement; in Fig. 286 the pressure rose as death approached; in Fig. 287 it fell.

The blood-pressure may be elevated for a time independently of arterial or renal disease.

Fig. 288 shows the blood-pressure in a man, aged thirty-eight, who was an iron-moulder by trade. He had always been healthy, save for an attack of pneumonia at the age of twenty-three, but had caught cold a fortnight before admission, though this had not kept him away from work. Ten days later he noticed that his eyelids were swollen in the morning, but the swelling disappeared as the day wore on; his back, he said, was sore when he stooped. He had a headache next day, and his feet were swollen at night. On admission, all these symptoms had lessened. There was a trifling albuminuria, and a little

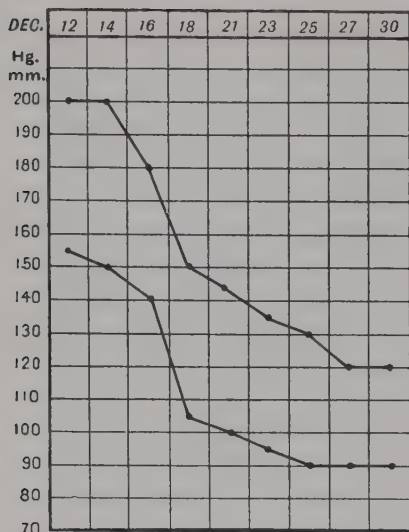


FIG. 284.—BLOOD-PRESSURE CHART.

Male, aged forty-seven: chronic nephritis, bronchitis; steady improvement.

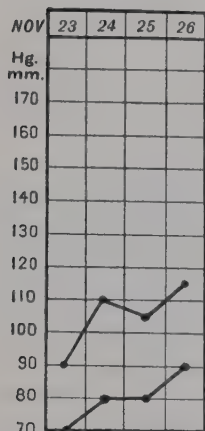


FIG. 286.—BLOOD-PRESSURE CHART, SHOWING SYSTOLIC AND DIASTOLIC PRESSURES.

Male, aged forty: alcoholism; pneumonia. Death on November 26th.

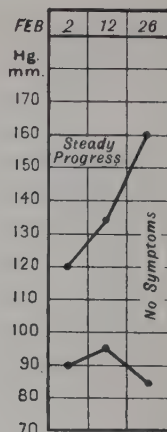


FIG. 285.—BLOOD-PRESSURE CHART.

Male, aged fifty-seven: aortic valvular disease; steady improvement.

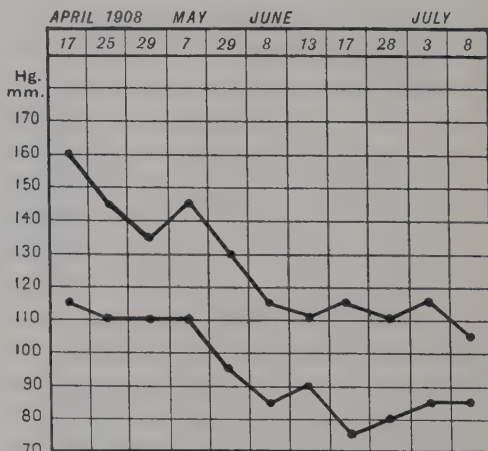


FIG. 287.—BLOOD-PRESSURE CHART, SHOWING SYSTOLIC AND DIASTOLIC PRESSURES.

Female, aged thirty-five; mitral disease. Death on July 23rd.

œdema in the legs below the knees, but the latter soon disappeared. The urinary output was always ample, and the albuminuria was trifling and sometimes absent. But it was eight days before the pressure fell within normal limits, and three weeks more before the minimum reading was recorded. There was no evidence otherwise of organic disease, and the elevation of blood-pressure was presumably toxic in origin.

High blood-pressure *per se* is thus no certain evidence of arterio-sclerosis, as it may be due to temporary causes, and obtains in some forms of valvular disease of the heart. In the absence of aortic incompetence, however, its indefinite persistence despite treatment, and the presence of hypertrophy of the left ventricle, are extremely suggestive of the disease. Widespread focal lesions in the thoracic aorta or the splanchnic arteries are the only other factors which can produce such a condition.

The apex impulse is an uncertain guide in some cases of arterio-sclerosis. It may be hidden by pulmonary emphysema, which not infrequently ensues in arterio-sclerotic cases. It may be weak, diffuse, and ill-sustained if cardiac failure has supervened; but, on the other hand, may still be punctate, powerful, and well-sustained even when œdema is widespread and general. In emphysema the cardiac sounds may often be heard best outside the left border of the cardiac dullness, even in the axillary line, thus indicating enlargement of the ventricle.

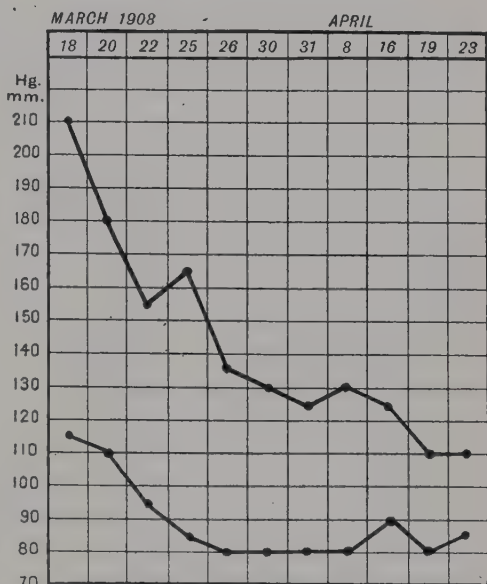


FIG. 288.—BLOOD-PRESSURE CHART, SHOWING SYSTOLIC AND DIASTOLIC PRESSURES.

Male, aged thirty-eight (*see* p. 503).

The cardiac sounds cannot be relied upon. The second aortic sound may be weakened, with failure, to such a degree as to be within normal limits, though it is often distinctly emphatic; or it may be wholly obscured by murmur. The special qualities of the first apical sound are often at fault, and it tells merely of mitral regurgitation or myocardial weakness.

If these data fail, the condition of the arteries may still be sufficient evidence, and it is here that ophthalmoscopic examination is so valuable, as changes are present in the large majority of cases, and persist irrespective of cardiac insufficiency (see Chapter XXX).

The presence or absence of albuminuria is of little value from the diagnostic point of view. Renal disease is a common accompaniment of arterio-sclerosis, but it may also occur independently. The small red kidney and the arterio-sclerotic kidney are usually associated with an albuminuria which is trifling in degree, and may even be wholly absent at any rate for a time, and the existence of considerable albuminuria indicates the presence of some new factor, such as passive venous congestion, or some accidental intoxication or toxæmia.

The symptoms which are met with in arterio-sclerosis are quite as varied as one would expect from consideration of the pathological lesions. Few of the viscera are absolutely normal, and any one of them may be specially damaged. But the kidneys are generally more or less involved, and renal excretion is frequently inadequate; and the heart, habitually working close to its maximum power, has a reserve which is but small, and in consequence readily exhausted; so that in practice *cardiac* and *renal symptoms* form the bulk of the complaints.

The symptoms which ensue on *cardiac failure* do not differ appreciably from those that may occur in cardiac insufficiency due to other causes; shortness of breath, palpitation or other cardiac discomforts, gastro-intestinal disturbances, œdema, etc., are the common complaints. In the earlier stages, when the failure is slight and relative, the diagnosis is easily made on routine examination; but in the later stages, and in the

presence of well-defined valvular flaws, one is apt to miss the underlying general condition.

Valvular flaws may arise in various ways. Arterio-sclerosis may hit a patient whose heart is already damaged by an acute endocarditis, or the converse may obtain. Arterio-sclerosis may cause local lesions in the arteries as well as in the viscera, and the valves may be deformed by a chronic degenerative lesion. The ventricle may give way before the strain, and dilate, with relative mitral incompetence. French writers are in the habit of emphasizing this difference in origin of mitral incompetence by the statement that in one group the lesion begins at the valves and ends at the myocardium, while in the other it begins in the myocardium and ends at the valves.

In uncomplicated cases of mitral disease the sequel of rheumatic endocarditis, the blood-pressure is uniformly normal or below normal. It is commonly asserted that the presence of œdema causes variations in the blood-pressure readings from the pressure exercised upon the capillary network in the tissues, but our experience is not consonant with this conclusion. The presence of œdema in the arms, however, interfering with the proper apposition of the armlet or the correct appreciation of the radial pulse, of necessity introduces factors which are likely to prevent accurate records; but in

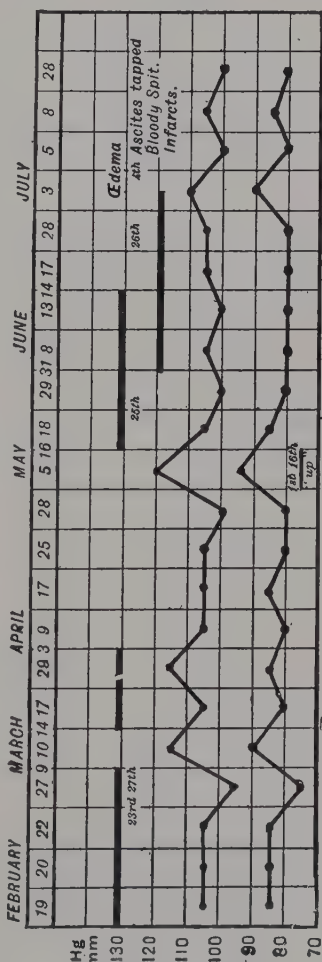


FIG. 289.—BLOOD-PRESSURE CHART.

Female, aged thirty: mitral valvular disease.

its absence it is astonishing to find how little variation occurs even in the presence of notable symptoms. In Fig. 289, for example, the systolic pressure only varied from 120 to 95 mm. Hg. during the course of observations extending over nearly six months. The patient, who was aged thirty, was admitted with orthopnœa, dropsy invading the trunk, ascites, and much catarrh in the chest. For a time all these symptoms were in abeyance, and she was out of bed and walking about the ward without discomfort, but the improvement was only temporary. She spat blood for over a month from pulmonary

infarcts, and ultimately left hospital during a recurrence of the symptoms. The highest readings were obtained at a time when compensation was fairly well established.

But other mitral cases occur with symptoms of a similar kind in which the blood-pressure is elevated, sometimes greatly, above the normal. This is shown in Fig. 290, which shows the pressure in a patient, aged fifty, who was admitted to hospital with orthopnœa, dropsy and ascites, in whom, with improvement, the pressure rose to even higher levels.

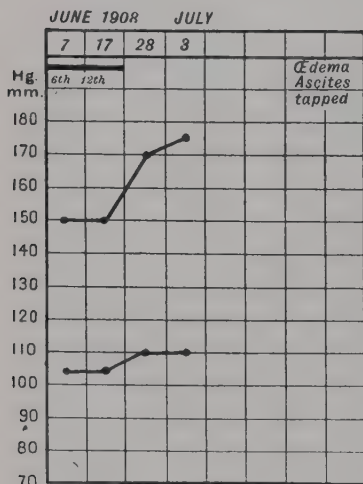


FIG. 290.—BLOOD-PRESSURE CHART.

Female, aged fifty: mitral disease
cirrhotic kidney.

It is extremely important to distinguish between these two groups of cases. In aortic stenosis, and in both the mitral lesions, the blood-pressure is not elevated to any excessive degree, no matter how large the left ventricle may be. The hypertrophy or dilatation is exactly calculated to compensate the valvular flaw, to ensure the entrance into the aorta of the normal amount of blood, and to maintain the aortic blood-pressure at normal levels. An intrinsic cardiac lesion (excluding aortic incompetence, the effects of which have already been discussed) is incapable of elevating the aortic pressure and the

presence of a high blood-pressure indicates the presence of some extrinsic influence—an intoxication, renal disease, or arterio-sclerosis.

The converse proposition is not, however, equally true. As one would expect, an elevated blood-pressure may fall to, or even below, normal from mere failure of the *vis a tergo*. The point is exemplified by Fig. 291.

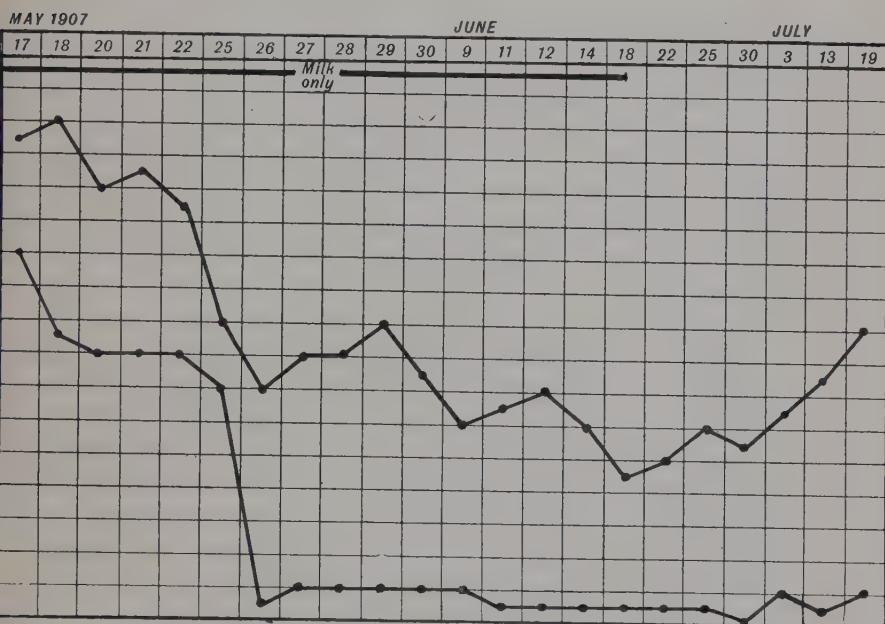


FIG. 291.—BLOOD-PRESSURE CHART.
Male, aged thirty-six: chronic nephritis.

The patient, who was suffering from chronic nephritis, was admitted complaining of headache, vomiting, and dimness of vision. The radial arteries were thickened, the left ventricle was enlarged, and the ophthalmoscopic picture was indicative of widespread vascular disease, and included several examples of miliary aneurysm. He was kept on a diet of milk alone from May 14th to June 18th, in the hope of allaying the renal mischief, and during this period the pressure steadily fell, ultimately reaching normal or perhaps sub-normal levels. But it rose again on the resumption of a more ample dietary, and at the time of his dismissal was distinctly high. His symptoms were in complete abeyance, and he was able to take a fair amount of exercise. It seems clear that, in this case, the blood-pressure required to be above normal

on account of the renal and arterial disease, and that the normal pressure on June 18th was due to cardiac weakness from relative starvation. There were no symptoms indicative of cardiac failure at this time, but he was, of course, confined to bed, and they would, we suspect, have appeared if he had been allowed out of bed. He again came under observation in October, with similar symptoms, and a systolic blood-pressure of 225-180 mm. Hg. Treatment was of little avail, and he died of uræmia shortly afterwards.

In those cases in which arterio-sclerosis and valvular flaws co-exist, a correct diagnosis may only be possible from observation of the arteries, the pulse, and the fundus of the eye. The cardiac evidence may wholly fail. The second aortic sound may be weakened from stiffness of the cusps, or replaced by murmur, and the first apical sound and the character of the apex impulse may be useless as guides. It should be remembered, however, that in uncomplicated mitral disease the second aortic sound is never accentuated, the pulse is never hard or well sustained, and that, with failure, the apex impulse becomes weak, diffuse, and of short duration.

The lesser *uræmic manifestations* are more likely to escape recognition than the serious symptoms, for the latter are usually associated with well-marked evidences of both cardiac and renal disease; but the diagnosis is generally definite on a careful routine examination. The symptoms not infrequently follow some special additional intoxication, often due to a gastro-intestinal upset, and may remain for long in abeyance if circumstances permit a mode of living on physiological lines.

Shortness of breath is a symptom of which arterio-sclerotic patients frequently complain, and requires some special attention, as it may arise in several ways.

Emphysema not infrequently occurs in middle-aged people from causes which are apparently trivial in their character. A cough, which is not necessarily of long duration or of great intensity, and may never have seriously inconvenienced the patient, may be the only apparent cause. But it will often be found, in such cases, that the pulse is hard, the arteries are thick, and the second aortic sound is accentuated. The lesion has resulted from apparently trivial causes by reason of the impaired nutrition produced by the arterial disease. *Emphysema* is often combined with more or less chronic bronchitis,

which may produce symptoms quite out of proportion to the intensity of the catarrh.

Both of these points were emphasized recently in the wards. Three men were admitted at the same time, suffering from bronchitis. One, aged forty-six, had been liable to bronchitis for many years, and on this account had been able to do but little work during the three preceding winters. His chest, as one would expect, was notably emphysematous. His heart and blood-pressure were normal. Another, aged fifty-nine, who was almost equally emphysematous, had never, he said, been off work previously from ill-health, and had never previously suffered from bronchitis. His left heart was considerably enlarged, and his blood-pressure, when the symptoms had abated, measured 175 mm. Hg. The third patient, aged thirty-eight, had no emphysema, and the pulmonary symptoms were not grave; but general œdema was so extreme that micturition was difficult from the tumefaction of the prepuce and scrotum. Some years before he had suffered from an acute nephritis, and, although the renal symptoms were trivial, the apex impulse was now $4\frac{1}{2}$ inches distant from mid-sternum.

In another group of cases shortness of breath is related to exertion, and is evidently due to *relative cardiac insufficiency*, even though the pulse is full and hard, and the left ventricle hypertrophied and powerful.

But in a third group of cases the dyspnoea is probably *toxic* in origin. Cheyne-Stokes respiration, for example, occurs almost exclusively in cases of nephritis and the "senile" type of cardiac disease, and is then present in the class of case in which elimination is likely to be insufficient; while it is conspicuously absent in the cardiac failure which accompanies acute endocarditis. Cardiac "asthma," too, is less common in the latter group than in the former, and primary asthma, as is well known, is not infrequently dependent on causes of a toxic nature. The slighter manifestations of dyspnoea sometimes own a similar origin, and eliminative treatment should always be pursued whenever dyspnoea is nocturnal or paroxysmal in character, or of a severity which seems disproportionate to the other evidences of cardiac or pulmonary embarrassment.

It is impossible to enumerate all the symptoms which may emerge in arterial disease.

They are frequently *cerebral*. In some instances their onset is sudden and fulminant, a cerebral hæmorrhage occurring without warning in a patient apparently in the best of health. But not infrequently minor manifestations of cerebral disturbance have been present for months prior to the stroke. An old man, who eventually died of cerebral hæmorrhage, appeared one day with a black eye, the result of a stumble over a stool. A few months later he missed the step of his carriage when entering it and again fell and injured himself. It was only on his third accident, when he again "tripped over a stool," that it was recognized that no stool was in the way, and that the fall was due to cerebral causes.

Headache is not uncommon. A man of fifty who had never had any serious illness before a gross stroke stated that for two years before this ensued he had frequently suffered from headaches, which were so severe at night that he was often unable to take any food when he came home and had perforce to go to bed at once. Some of these patients complain of giddiness and faintness, particularly upon sudden changes in position, such as when getting out of bed in the morning, or rising from the kneeling posture.

In another group the symptoms are referable to the *extremities*, complaint being made of numbness, tingling, or coldness. Cramps sometimes occur, even of such severity as to prevent sleep. The intermittent claudication, first described as occurring in horses, obtains also in man, pain of an excruciating character occurring in the legs upon exertion, and necessitating its instant cessation. After a few minutes' rest the pain lessens and disappears, to recur, however, whenever an effort of equal intensity is again made. In other cases the lower limbs feel heavy, though not painful. Thus, in one of our patients, a man aged sixty-nine, suffering from angina pectoris, the first symptom complained of was inability to drag one leg after the other if he had walked for more than about 150 yards. After a rest he was able to resume his walk for another 100 or 200 yards, but then he had to rest again. In this manner, alternately walking and resting, he could cover a distance of a mile or more.

Gastro-intestinal discomforts are not uncommon, nausea and discomfort after food, and flatulence both gastric and intestinal. These may be absent when existence is placid and unruffled, and may only occur after a hearty meal, or one taken when cold or tired.

Hæmorrhages occur in other organs than the brain. The epistaxis of elderly people, for instance, if not associated with an active lesion in the nose, is generally the result of arterial disease; and bleeding of a like character elsewhere, from the lungs, the stomach, the bowel, or the bladder, may be the source of an idiopathic and temporary, but often recurring, hæmorrhage.

It must be recognized that not infrequently in middle-aged and elderly people there is, underlying any local symptom which may require attention, a widespread general disease—arterio-sclerosis—a disease which is essentially progressive, though its progress may be delayed by suitable treatment; and in which, quite apart from any special lesion, the arteries are more or less damaged, the cardiac reserve is lessened, and the renal functions are more or less impaired.

CHAPTER XXX

THE OCULAR MANIFESTATIONS IN ARTERIAL DISEASE

BY ARTHUR J. BALLANTYNE, M.D.

THE portion of the central artery of the retina contained within the optic nerve has the same structure as any other artery of similar calibre. When it reaches the lamina cribrosa, however, the membrane of Henle disappears, and is replaced by a fine network of interlacing elastic fibres between the endothelium and the outer coats. In the smaller arterial branches in the retina itself these fibres become scanty, and in the smallest twigs they disappear altogether. The central retinal vein behind the lamina cribrosa has no membrane of Henle. Its place is taken by abundant fine interlacing elastic fibres, which, however, are absent from the venous branches in front of the lamina cribrosa. The vein contains no muscular fibres.

In their normal state the walls of the retinal vessels are so transparent that what we see on ophthalmoscopic examination is merely the blood column. Reflection of light from the convex anterior surface of this column produces, in the arteries, the appearance of a continuous light streak along the centre of the vessel, its width being uniform, and equal to about one-third of the apparent width of the artery. In the retinal veins this central streak is relatively narrower, and is not so continuous as that of the arteries.

The earliest histological changes in retinal angio-sclerosis are intimal, but these are soon followed by changes in the outer coats, and the early clinical diagnosis undoubtedly rests

mainly on the discovery of changes in the calibre of the vessels due to intimal thickening, and alterations of the transparency of their walls due to proliferation and degeneration in their outer coats.

In the great majority of cases the first ophthalmoscopic evidence of angio-sclerosis in the retina is a *reduction in the calibre of the arteries*, due to thickenings of the intima. At first these thickenings affect only short segments of the arteries, and produce the appearance of localized constrictions, or, if constrictions succeed each other at short intervals along the course of the vessel, the resulting appearance is that of a chain of spindle-shaped segments. Sometimes, instead of local thickenings of the intima there is a more widespread change which produces a *uniform narrowing* of the arterial lumen, so that the retinal arteries as a whole may be apparently reduced to mere threads.

Associated with these early changes in the arteries, we can usually observe *constrictions of the retinal veins* where they are crossed by the arteries. This is apparently not due to any change in the vein wall, but merely to the pressure of the more rigid overlying artery. That this is so is suggested by the fact that the vein looks broader on the distal side of the constriction, as if from mechanical stasis of the venous blood-stream.

Two other conditions which are often present about the same period, namely undue *tortuosity of the arteries* and an *excessive brightness of the central light streak*, the latter giving to the artery the so-called "*silver wire*" appearance, are to be looked upon, not as evidences of retinal angio-sclerosis, but as accompaniments of excessively high blood-pressure. They are most frequently seen in cases of chronic renal disease, especially of granular contracted kidney.

Miliary aneurysms, in the form of small globular or fusiform dilatations of the arteries, may also be seen with the ophthalmoscope. Histologically, such dilatations have been observed still more frequently on capillaries and other vessels of microscopic size, and no doubt they form weak points in the vessel walls from which hæmorrhages may occur.

It has already been stated that the walls of normal retinal

vessels are invisible, so that what we see with the ophthalmoscope is merely the blood column. The more advanced stages of arterio-sclerosis render the vessel walls themselves visible. The first evidence of *loss of transparency of the wall* is that the blood column looks paler, and the central light streak less striking than normal. At a later stage the process of thickening and degeneration in the vessel wall proceeds so far as to produce the ophthalmoscopic picture known as "*perivasculitis*," the blood column being bounded on each side by a clear white line, or entirely concealed, for a smaller or greater part of its course, by a dense white band, which represents the opaque wall of the artery. This appearance does not imply the complete obliteration of the lumen, and the conversion of the artery into a solid strand of tissue. The opaque wall often conceals a blood column, which, however, may be extremely narrow.

Histological examination shows that the changes corresponding to these ophthalmoscopic appearances consist of, first, proliferation of the elastic and other fibrous elements of the vessel wall and, later, degeneration, mainly hyaline, of the same tissues. If, as is usually the case, white bands such as those described are due to sclerosis of the medial and adventitial coats, the total width of the white band will not be appreciably greater than that of the original blood column; but occasionally we see a broader white band or patch, not associated with narrowing of the neighbouring blood column, which is probably brought about by cellular infiltration, followed by degeneration, in the perivascular sheath.

Bright, glistening spots are occasionally seen in the walls of the blood vessels, and these may represent deposits of lime or of cholesterine.

The veins may, but do not usually, show changes as marked as those in the arteries. *Constriction* of the veins where crossed by the arteries has already been mentioned. In the more advanced stages the veins become *dilated and tortuous*, and may show *irregularities of calibre*, or beading, similar to those seen in the arteries. *Aneurysmal dilatations* are also sometimes seen on the veins. Excessive prominence and tortuosity of the finer venous radicles is rather a char-

acteristic appearance, especially found in association with advanced degenerative renal disease.

In some cases of retinal angio-sclerosis *congestion of the optic nerve and œdema of its margin* and of the neighbouring retina is observed. *Retinal hæmorrhages* are another common accompaniment, and may be present even when the only visible changes in the vessels are the early alterations of arterial calibre, and with or without hæmorrhages we may see white spots and patches due to degeneration of retinal elements. These changes, often described as a variety of albuminuric retinitis, may be found even in the absence of renal disease, and it is indeed uncertain how far the characteristic changes of albuminuric retinitis are due to local vascular changes, and how far both are traceable to the one underlying cause.

Sclerosis of the larger choroidal vessels frequently occurs, and when the overlying tissues are sufficiently transparent to render them visible, they present the appearance of an irregular network of white lines.

Two conditions which present very striking and characteristic pictures may be referred to as strong evidence of the existence of local vascular disease. These are: *Obstruction of the central artery* and obstruction of the central vein of the retina. In the former the retina over the whole of the posterior part of the fundus is the seat of a diffuse, milky white opacity, in the centre of which the fovea stands out as a bright cherry-red spot. The disc is pale, the arteries are reduced to threads, and the veins are also somewhat narrow. This is the picture which used to be considered peculiar to embolism of the central artery, but it has been proved that in many cases the obstruction is the result of local endarteritis or endarteritis *plus* thrombosis. In such cases it is the rule to find other evidence of local endarteritis in the shape of irregularity of arterial calibre, either in the retina of both eyes or in that of the affected eye only.

Obstruction of the central vein of the retina is usually caused by thrombosis associated with local disease of the vessel wall. On ophthalmoscopic examination the veins are seen to be dark, distended and tortuous; the arteries are narrow, the nerve-head is congested and œdematous, and the retina

contains abundant hæmorrhages and white exudative patches. This condition is apt to be followed by the development of secondary glaucoma or proliferative retinitis.

Other conditions might be mentioned here which are evidence of the presence of local arterial disease—retinitis circinata, retinitis with massive exudate, etc.,—but these are among the rarer ophthalmoscopic findings, and have not so direct a bearing on the state of the general vascular system.

The significance of these various retinal manifestations of angio-sclerosis lies in the close correspondence which has been found to exist between the retinal vessels and cerebral vessels of similar size. They resemble each other histologically, and there is a good deal of similarity in the physical environment of the retinal vessels under intra-ocular pressure and that of the cerebral vessels under intracranial pressure. There is no physical sign, e.g. the height of the blood-pressure, the pulse, the condition of the urine, or even the state of the heart and peripheral arteries, which gives such a reliable index of the probable state of the cerebral circulation as does the appearance of the retinal vessels. Moreover, as has been so frequently pointed out, there is no other region of the body where direct examination of the living blood vessel can be so satisfactory and complete as in the case of the retina, and the detection of the retinal hæmorrhages and exudates which may accompany vascular changes is a further valuable aid to the diagnosis and prognosis of the patient's general state.

That there is an important relationship between pathological states of the retinal and cerebral vessels has long been clearly recognized. It would be difficult or impossible to express this relationship with such precision as to say what percentage of cases of cerebral disease are the subjects of retinal vascular changes, or *vice versa*; but every oculist is familiar with the main fact of the case from the ophthalmic point of view, namely, that a large proportion of the patients in whom he discovers retinal angio-sclerosis suffer from symptoms of cerebral angio-sclerosis, and become the victims, at a later date, of cerebral hæmorrhage, thrombosis or embolism.



FIG. 292.—RIGHT FUNDUS OCULI FROM A CASE OF CHRONIC NEPHRITIS.

A. J. B. *del.* See p. 519.

No doubt a solitary retinal hæmorrhage may occur, just as a conjunctival ecchymosis may, without any discoverable cause in the general circulation, but the presence of even one or two small retinal hæmorrhages naturally demands careful investigation of the state of the heart, kidneys and blood vessels, and in this investigation the state of the blood-pressure is probably of much greater significance than the presence or absence of albuminuria.

If retinal hæmorrhages are associated with some of the signs of arterial disease which have been described, there is a strong presumption that the smaller cerebral vessels are in an unhealthy state, and that there is a real danger of a cerebral accident of some kind. It is not wise, or even possible, to estimate the probable duration of life on the basis of the fundus changes, for in some cases the end comes with surprising rapidity, while other patients live for many years after the first discovery of the arterial disease. Much depends on the patient's environment, occupation and means, so that the prognosis is naturally more grave in a labouring man, who works under unfavourable conditions as to hours, weather and periods of rest, than in a man in comfortable circumstances, who can make the care of his health his first concern.

The *prognosis* is worse the younger the subject, and in the young, too, the presence of albuminuria adds to the gravity of the prognosis more than it does in the elderly. The presence of glycosuria is an additional source of anxiety, and other factors which increase the seriousness of the prognosis are a personal history of chronic alcoholism and a family history of cerebral vascular disease.

Note.—The accompanying plate illustrates many of the vascular conditions referred to above. It represents the right fundus oculi of a man suffering from chronic renal disease. A certain indistinctness of the disc margin, due to œdema, which was also present, is not shown in the drawing. The lower temporal artery is unduly tortuous, and presents the "silver wire" appearance referred to. Three months before this drawing was made three or four small aneurysms were present on this artery where the white spots are now seen. A branch which leaves the same artery nearer the disc passes out across the fundus as a white opaque streak (*periarteritis*). The upper nasal artery shows sudden and marked reduction of calibre a short distance

from the disc (*endarteritis*). The veins on the whole are distended, and are seen to be constricted where they are crossed by the arteries. The fine terminal twigs are abnormally distended and tortuous. In the lower inner region of the fundus one of these twigs shows something resembling an aneurysmal dilatation. The retinal hæmorrhages and white spots were much larger and more numerous at an earlier stage of the disease.

CHAPTER XXXI

THE TREATMENT OF ARTERIO-SCLEROSIS

THE treatment of arterio-sclerosis is essentially preventive, and thus lies to the hand of the medical attendant. The disease is very chronic, and one in which, as a rule, the early symptoms are trivial, though serious symptoms may develop suddenly; and even when the slighter manifestations are equivocal, the underlying condition is generally apparent *when it is looked for*. And if, as we believe, Sir Clifford Allbutt's theory of its origin is applicable to a large number of cases, its development may be prevented, or at any rate its progress may be retarded to a degree which as yet seems scarcely to be appreciated. But, as Huchard well said, the treatment is a *régime*, and not a drug, and a *régime* which may be impossible for a busy man to follow; and in such cases we can but deliver to our patients the ultimatum—a radical change of habits or a short life—and abide by the decision of the person most concerned.

The introduction of the Riva-Rocci sphygmomanometer into clinical work naturally evoked the hope that the blood-pressure chart would supply more definite indications for treatment than had hitherto been available, but a very short experience sufficed to show that this was erroneous, and that it was *per se* an unreliable guide. On reflection, too, the fallacy was obvious. In acute nephritis the blood-pressure is elevated in the presence of symptoms, and returns to normal long before the albuminuria has disappeared. But in those forms of chronic nephritis with induration, the small white and the small red kidney, the blood-pressure, even in the absence of symptoms, never runs at a normal level. Structural changes have occurred, and the arteries are *permanently* thickened; the heart is *permanently* enlarged, and the blood-

pressure is *permanently* elevated. And just as the amount of albumin in the urine is an unreliable guide to the functional activity of the kidneys, so the height of the blood-pressure fails to reveal the degree of damage that has been done to the arteries and to the heart. In arterio-sclerotic patients whose elimination is even relatively insufficient, the blood-pressure varies considerably from apparently trivial causes; a mere bronchial or gastro-intestinal catarrh may suffice to elevate it, cardiac failure may lower it, and every nicety of examination and the most careful survey of the case may for a time prove incapable of solving the important question of the height of blood-pressure which must now be considered "normal" to the particular individual. A systolic reading of 180 mm. Hg. may be too low for perfect elimination, or too high for the heart to cope with successfully.

Notwithstanding these strictures, routine estimation of the blood-pressure and a daily chart are of great value in guiding the treatment of the case. Isolated observations are of little use, particularly those taken in the consulting-room, where so many factors (emotion, fatigue, etc.) may be in action, producing a variation from the average reading. And the blood-pressure chart by itself is equally unreliable, and is only helpful when it is taken into consideration along with those other data which are usually available.¹

In the early stages of the disease it is often difficult to relate the trifling symptoms to their exciting cause, and as it is more easy to discern the process in cases where the symptoms are well marked, the latter will be considered first.

The onset of serious symptoms is as a rule *more or less abrupt, and the result of the operation of some new factor*. *A priori*, it might be thought that the onset would be gradual

¹ The systolic blood-pressure in healthy persons is lowest in early life and increases steadily as years go by. In the second decade it is usually about 110 mm. Hg., in the third about 120 mm., in the fourth about 130 mm., and in the fifth about 140 mm. A constant pressure of over 150 mm. is probably always a sign of a pathological condition. There is, however, considerable difference in individuals, and a pressure of 130 mm., or thereabouts, may never be exceeded even in later life. The diastolic pressure runs 20-40 mm. below the systolic.

in response to the steady increase in the grade of the arterial lesions or the renal inadequacy, or the gradual diminution of the cardiac reserve; but such a course, though it may occur, is distinctly unusual. The minor symptoms occur insidiously, but the individual is curiously tolerant of visceral weaknesses of gradual onset, and instinctively learns to protect himself from his increasing disabilities. Business responsibilities are turned on to younger shoulders, the "single" game of golf is replaced by the "four-some," alcohol and tobacco are thrown overboard or their consumption is restricted, and a quiet life with little sudden strain permits the overtaxed organs to perform their work with but small inconvenience to their owner. There is, however, little or no reserve, and serious symptoms result from conditions which, in a healthy individual, would occasion no anxiety.

Bronchitis is a common cause of trouble. Chronic bronchitis is a very frequent accompaniment of arterio-sclerosis, and an exacerbation of the catarrh may produce symptoms which are quite out of proportion to the intensity of the pulmonary lesions. A man of forty-seven years, a stone-breaker by occupation and an alcoholic by choice, contracted a cold, to which he at first paid little attention. A week later he had become very short of breath, and his feet were swollen. When he was admitted to hospital a fortnight after the onset (Fig. 293), œdema was universal and considerable. There were copious albuminuria and slight hæmaturia, and a well-marked bronchitis. Within the next ten days the œdema disappeared, and he lost 1 stone 10½ pounds in weight,

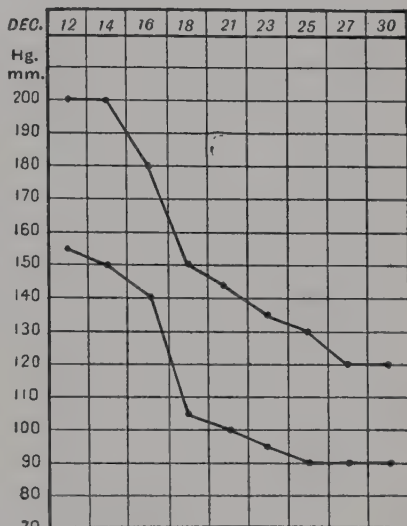


FIG. 293.—BLOOD-PRESSURE CHART.

Male, aged forty-seven: chronic nephritis; bronchitis; improvement.

the albuminuria became trivial, and the bronchitis subsided. His arteries were tortuous and but slightly thickened. The systolic blood-pressure, on admission 200 mm. Hg., fell steadily during convalescence, and soon reached sub-normal registers.

The illness in this case seemed wholly attributable to the bronchitis, which was, however, associated with symptoms which are not its usual accompaniments. The rapid improvement in the renal symptoms negatived the diagnosis of an

acute nephritis, and suggested the irritation of an already damaged kidney, an inference which was strengthened by the presence of arterial thickening and a white patch in the retina of the right eye.

Evidence of cardiac failure may be well marked in these cases. A man, aged sixty-six, who was habitually intemperate and suffered from a chronic cough, contracted a fresh bronchitis. He became very short of breath, and had to be propped up in bed at night, and suffered from severe pain in the epigastrium. Within a week he was forced to cease work and to seek admission to hospital. He was now notably cyanosed and very breathless, requiring to sit almost upright in bed. The bronchitis was

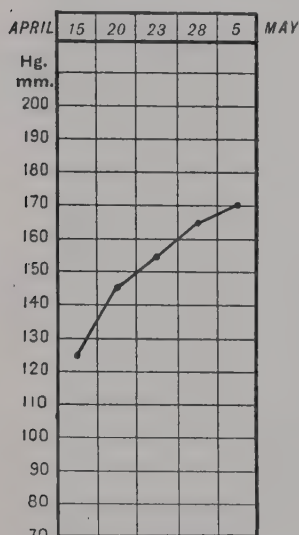


FIG. 294.—BLOOD-PRESSURE CHART.

Male, aged sixty-six :
bronchitis.

widespread, and the liver was enlarged and tender; the feet were slightly swollen, and the mitral valve was incompetent. The urine contained a trace of albumin. Improvement was rapid, and he was soon lying flat in bed, while the apical murmur and the enlargement of the liver had disappeared. Coincidentally the systolic blood-pressure rose from 125 to 170 mm. Hg. (Fig. 294). The bronchitis had evidently put a strain upon an already overtaxed heart which it was unable to overtake, and the consequent failure was severe and alarming.

Excessive indulgence in *alcohol* may produce serious symptoms. A tailor, aged fifty-one, had enjoyed good health prior to the onset of the symptoms for which he sought advice, save for two attacks of renal disease, one at the age of twenty-five, following scarlatina, the second of obscure origin at the age of forty-one, from both of which he had made good recoveries. One day he took drink to excess. He went to his work next morning, but about three o'clock in the afternoon was noticed to be somewhat stupid. He walked to the door to get some fresh air, and soon after his return to the workshop became unconscious and had a short general convulsion, from which he rapidly recovered. His systolic blood-pressure was 220, the diastolic 145 mm. Hg. Six weeks later he again got drunk, and again had a convulsion; and a fortnight later another fit occurred, this time apart from alcoholic excess. He subsequently became fairly temperate, and the convulsions did not recur, but he "aged" rapidly and became less fit for work. Two years later headaches ensued, and he had to cease work, and in a few months succumbed to an apoplectic seizure associated with hemiplegia.

Sepsis is another, but less frequent, cause. A man, aged fifty-two, who had probably suffered from syphilis, and had had an attack of nephritis at the age of forty-six, tore the instep of his foot with a rough boot-eyelet. The wound suppurated, and was poulticed. A week later the other foot was found to be swollen. In a day or two the eyelids became baggy, and he complained of headache and of frequency of micturition. He was very breathless and had to sit upright in bed. A similar origin was observed in a man, aged seventy-five, whose increasing difficulties had been carefully watched for some years. A not very large gum-boil formed about a carious tooth, and he was fevered for twenty-four hours until it was evacuated. But general symptoms ensued, and he became drowsy and mildly delirious and extremely weak, and the symptoms only subsided slowly. The minor forms of sepsis, particularly those associated with carious teeth, are probably of more importance in this respect than is generally realized.

Symptoms not infrequently succeed severe *hæmorrhage*,

whether gastric or intestinal, or, as in the following case, occurring *post-partum*. A woman, aged thirty-five, who had always been healthy save for an attack of rheumatic fever at the age of twenty, which kept her in bed for three months, noticed that her feet were swollen in the latter half of her fourth pregnancy. Labour was difficult and prolonged (twins), and she lost much blood. The œdema, however, rapidly disappeared, and she felt quite well when she got up out of bed on the ninth day, but it soon recurred and became extreme. She was now (Fig. 295) very short of breath, with a troublesome cough and expectoration; the bowels

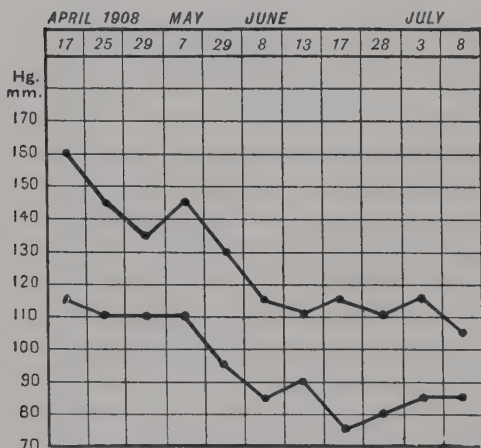


FIG. 295.—BLOOD-PRESSURE CHART.

Female, aged thirty-five: mitral disease; cirrhotic kidney. Death on July 23rd.

were loose, and she felt sick and vomited on several occasions. The symptoms steadily increased in severity, and she died three months later. The strain of pregnancy and the debility consequent on the hæmorrhage proved too great for a heart which had little reserve power.

Starvation often produces notable symptoms. A woman, aged sixty

years, who was in extremely poor circumstances, and who had been starving for some weeks, noticed that her feet had become swollen. The œdema rapidly involved the trunk and face, and she was forced to stay in bed. She felt sick, vomited repeatedly, and was very short of breath. The striking feature in her condition on admission to hospital was the emaciation. The muscles were tiny and soft, the skin was dry and wrinkled, and the subcutaneous fat was minimal. In notable contrast thereto, the pulse was full and hard, the systolic blood-pressure being 190 mm., and the diastolic pressure 135 mm. Hg. (Fig. 296). The symptoms

subsided as her general nutrition improved, but the blood-pressure remained high.

One is apt to forget when treating a patient with a local disease that the symptoms may be due to a general condition and not to the local lesion, although both the prognosis and the treatment depend upon the correct appreciation of the factors which are producing the symptoms at the particular moment. When symptoms arise insidiously the outlook is, as a rule, more serious than when they occur as the result of some additional new factor, for the lesions are usually already advanced. In a general way the immediate prognosis in private practice is worse than in hospital patients, for in the former the conditions of life are more satisfactory and the breaking strain has been but slight. If immediate recovery ensues, however, the prognosis in private patients is generally the better, for a reasonable life of leisure is usually unattainable among the older hospital patients. Their financial position, for instance, is rarely comfortable, and their alternative lies between continuance of labour and at any rate relative starvation and discomfort; so that it is generally impossible for the first and most important indication for treatment—the removal of the cause which induced the occurrence of symptoms, and the prevention of its repetition—to be followed.

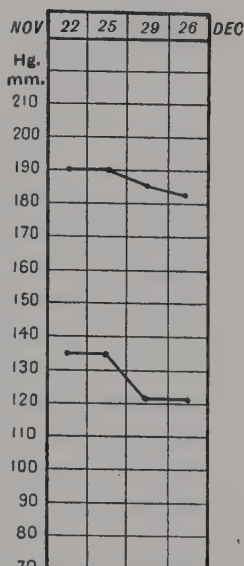


FIG. 296.—BLOOD-PRESSURE CHART.

Female, aged sixty :
cirrhotic kidney.

The treatment of arterio-sclerosis in the early stages, when the symptoms are obscure and slight, is most important, for it is then that its effect is greatest, not only in the relief of symptoms, but in arresting the progress of the disease.

The general indications have already been laid down. The blood-pressure is elevated by the presence of various deleterious substances in the blood. Continued high pressure produces permanent structural alterations in the heart and

blood vessels—arterio-sclerosis. In every case of arterio-sclerosis renal elimination is more or less insufficient, the cardiac reserve is lessened, the viscera are poorly nourished, and their functions are imperfectly discharged.

There is a general consensus of opinion that a generous diet and excessive consumption of alcohol lead in time to "plethora" and increased arterial pressure. Sir Clifford Allbutt has crystallized this in his statement that the most important cause is "repletion, relative or positive—*i.e.*, tempered or untempered by air and exercise," hindering the capillary flow, and augmenting the arterial blood-pressure. It seems probable that Sir Clifford Allbutt's theory is applicable to many cases, the type of which is the big, burly individual, energetic in all his habits, and overburdened with adipose tissue. But the disease is not uncommon in quite another type, under average in size and weight, of sedentary habit, and from circumstances or choice exceeding neither in eating nor drinking. William Russell has suggested the probable explanation. It is not the *quantity* of the food that is at fault in these patients, but *imperfect digestion*. The primary error may be very varied, an injudicious choice of food, gastric, intestinal, hepatic, or pancreatic insufficiency; but the ultimate result is the same, imperfect and disturbed metabolism and persistent intoxication. Further, we remember that amines may be formed from amino acids by bacterial action during the metabolism of protein, and that while histamine lowers the blood-pressure, tyramine raises it.

The kidneys are mainly concerned with the removal of the waste products of protein metabolism. If this is excessive, their functions may be overtaxed; if it is abnormal, abnormal substances may be formed and excreted in the urine (*e.g.*, sugar, indican, etc.), and there is evidence that most of these bodies irritate the kidneys during their excretion. Under normal conditions, carbohydrate and fat metabolism impose little work upon the kidneys, unless their consumption be excessive (*e.g.*, alimentary glycosuria), but in disease their metabolism may be imperfect, and the products may be excreted in the urine (*e.g.*, sugar, acetone series, oxalates, etc.).

In a general way the intake of food must be carefully super-

vised and excess avoided, and a minimum of protein food allowed. But the proverb, "One man's meat is another man's poison," is peculiarly applicable to arterio-sclerotic patients, and a failure of digestion and the consequent irritation of the tissues from the presence of injurious substances in the blood may lead to the occurrence of serious symptoms.

Appetite, for instance, is essential to perfect digestion, and may fail before the "simple life and food." Vegetarian diet is well recognized to be difficult of digestion, and the glass of wine at meals, or savoury dishes, may be *necessary* stimulants for the individual. If digestion is imperfect, the undigested foodstuffs in the bowel may ferment or putrefy, and a new series of irritant materials may become available for absorption. As a result of "fermentative" processes, acetic, lactic, propionic, butyric acids, etc., may be formed; while from protein "putrefaction," neurin, cholin, putrescin, cadaverin, mercaptan, H_2S , phenol, cresol, skatol, indol, etc., may be produced.

This point does not seem to have been sufficiently recognized, perhaps because the effects of indigestion vary so greatly even when the digestive disturbances are apparently comparable. But in one patient a bout of vomiting or an attack of diarrhoea may rapidly remove the irritant, while in another headache, depression, irritability, etc., show that absorption has occurred. There is no exact relationship between the amount available for absorption and that actually absorbed; for, as Herter has shown, indicanuria may be well marked when the faecal indol is inconsiderable, and insignificant when the latter is excessive.

A suitable dietary, then, cannot be rigidly laid down without considerable experiment. It is important to make changes *slowly*, and to regard the general condition of the patient as a whole. One is apt to limit the amount of food too strictly, and the latter error may be as injurious as over-feeding. But no diet is suitable unless the tongue is clean, gastro-intestinal symptoms wholly absent, and the stools normal in colour and odour and consistence.

The "output" in these cases is quite as important as the "intake," and requires equal attention. A daily evacuation

of the bowels may, for instance, be insufficient, and we have more than once been able to feel the colon throughout its course, though the bowels moved regularly every day. In some of these cases regular laxative medicine may be required, in others only occasional doses; but the weekly "wash-out" is probably always salutary even when it seems unnecessary. The kind of drug employed is of less importance than the thoroughness of the process. Our preference is for a mercurial, in particular blue pill, with a saline as the occasional purge, and the milder salines, cascara, or senna, for regular use.

The kidneys, too, require regular flushing. A simple diuretic mixture is often helpful, but it should be subservient to the ingestion of water in amount, the daily output of urine being kept over 60 ounces. But here, again, care is necessary, for the "long" drink at meals is not unlikely to disturb digestion. Fluid should be taken *when the stomach is empty*, the first thing in the morning, a full half-hour before luncheon and dinner, and perhaps at bedtime.

The skin, too, must be kept active. A Turkish bath once or twice a week is often well borne, but regular toilet ablutions and mild sweating exercises are probably better; and many amusements may be utilized—golf, riding, tennis, shooting, fishing, etc.—if they are available, and of a degree adapted to the physique of the particular individual.

In the treatment of an individual patient it is generally wise to insist at the outset upon a strict *régime*. The details are thus better learnt, and as the symptoms remit, relaxations can readily be allowed, while an increase of the restrictions after a period of treatment is naturally disappointing to the patient.

In any case where the symptoms are severe, confinement to bed is generally advisable at first. The bowels should be kept freely open by salines and blue pill, the fluid intake increased so that 60 to 90 ounces of urine are passed *per diem*, and elimination assisted by a simple diuretic mixture. The skin should be kept active by a daily warm bath, and the diet restricted to three or four pints of milk, with or without the addition of some farinacea. Milk is usually well borne by

these patients ; if there is any digestive discomfort, it can be overcome by peptonizing or citrating the milk, or by giving it as curds or "soured." As a rule the symptoms rapidly subside as soon as excretion is ample. But restrictions of this kind cannot be maintained for long with benefit, and in the absence of symptoms some addition should be made to the diet after a few days. The quantity of farinacea is increased, and white fish and eggs are given in addition, but their amount must at first be limited and only gradually augmented. The cooking must be of the plainest kind, but every possible variation should be used, so as to encourage appetite and promote digestion. In the majority of cases improvement continues, and the diet may then be still further varied by the inclusion of chicken and rabbit and a little potato. The key to treatment at this stage is the condition of the digestive functions and the general well-being of the patient rather than the height of the blood-pressure or the amount of albumin in the urine.

Some digestive help may be required—some rhubarb or soda, or a pepsin mixture—and the tongue must be clean, the stools normal, and gastro-intestinal symptoms in abeyance before any notable addition to the diet is made. Digestive disturbances are not infrequently the result of oral wrong-doing, and every effort should be made at the outset to ensure that oral sepsis is absent, and that efficient mastication is possible ; and the patient's attention should be drawn to the necessity that it should be thorough and complete.

Mild massage may be commenced at this stage as a preliminary to a certain amount of exercise, and the latter, when permitted, must be supervised at first by the physician as sedulously as it is done in a sanatorium for tuberculous patients, for the general feeling of well-being, often coupled with a loss of superfluous fat, is apt to incline the patient to an amount of physical exertion which is too great for his flabby muscles.

There is always difficulty in regulating these details when a return to work is finally allowed, for the exigencies of business necessarily interfere with the placid, orderly routine of life which is so essential to an arterio-sclerotic patient. The

daily time-table must be laid down by the physician, and particular care taken that the meals are regular and of suitable quality ; and that some rest is possible both before and after them.

The diet in convalescence is often restricted within too narrow limits. The cause of the disease is often dietetic, and the error may arise in different ways. In one case it is the quantity of food that is at fault, in another the quality ; while in a third both of these factors may be satisfactory, and the error lies in faulty digestion. It is, of course, manifest that all foods which are notoriously difficult of digestion (crab, goose, etc.) or decomposing (high game) must be avoided, as well as all salted foods, for it is notorious that the excretion of salt is almost always defective. The red meats are usually interdicted. In a general way they are as easily digested as the white, and contain little more extractives ; but they are essentially the savoury foods, and so likely to encourage overeating ; and they are often spiced and salted in the cooking to a degree that must entail considerable strain on elimination. The ordinary *consommé*, for example, contains a minimum of nourishment and a maximum of substances to be excreted by the kidney, so that all soups made with meat stock should be prohibited. Vegetable stock, however, can be utilized.

A little roasted mutton or a lightly shot fresh young bird is, however, certainly permissible as an occasional variation. The viscera are, of course, objectionable on account of their purin content. The vegetables only require consideration from the digestive point of view, and the majority of the fruits can be allowed in their season.

The use of drugs in arterio-sclerosis requires some further consideration. *There is no specific medicine, and the essential part of the treatment is the regulation of the life of the patient on the lines that have been suggested.* But in arterio-sclerosis, as in other chronic diseases, various symptoms may arise from time to time, and the particular measures that are required for their relief are generally indicated by the special disorder.

In the latter stages the symptoms may be referable to the heart, the kidneys, or the vessels.

Cardiac failure produces symptoms similar to those which occur in chronic valvular disease, shortness of breath, œdema, etc., and in the main the comparison is exact. In some instances, however, a difference may be observed. A patient who is dying from the cardiac failure of pernicious anæmia as a rule lies flat in bed and is rarely orthopnœic, even when œdema is considerable and widespread. Cases of serious failure in valvular disease in early life may have notable distress in breathing, and may have to sit upright, but there is some adequate reason for the dyspnœa, bronchitis, infarct, or pleural effusion. But in arterio-sclerosis dyspnœa may be extreme and orthopnœa absolute without any obvious pulmonary lesion, though of course such a condition may be present, and then intensifies the distress. Paroxysmal dyspnœa, whether of the asthmatic or Cheyne-Stokes type, rarely, if ever, occurs in uncomplicated cardiac disease; its presence seems always to indicate imperfect elimination, as a rule of renal origin.

The chief difficulty in the treatment of these cardiac cases is the difficulty of recognizing the precise mode of the failure, for failure may occur in two ways. In one type elimination becomes insufficient, and the blood-pressure is in consequence elevated to a height at which the heart is unable to work easily. In the other, the cardiac failure is primary.

In acute nephritis, as is well known, the blood-pressure is higher than normal. With improvement and the disappearance of symptoms it returns to the normal as a rule long before the albumin has disappeared from the urine. In arterio-sclerosis, on the other hand, the blood-pressure is permanently elevated. If elimination is insufficient in arterio-sclerosis, as in acute nephritis, the blood-pressure rises, in the former case to extraordinary heights, and the heart may give way before the extra strain—a mode of failure which is, indeed, well known to occur occasionally in acute nephritis. But we do not know, in an elderly person with cardiac failure and high blood-pressure, *how much of the latter is due to permanent conditions and how much to temporary causes*, unless our observations have been continuous and antecedent to the onset of symptoms. In arterio-sclerosis the permanent elevation of pressure gradually becomes higher

as time rolls on, and in due course the heart will fail before its task, and the blood-pressure will fall, sometimes very considerably, with the onset of symptoms. To follow the well-known simile of the horse and cart of coals that has stopped on a hill, we are in doubt whether to apply the whip or to lighten the load so as to enable the height to be surmounted, and we have often to pursue—tentatively and with care—both courses.

The vagaries of blood-pressure charts are extremely curious,

and their interpretation may be impossible for a time.

A baker, aged thirty-eight, was admitted to hospital, complaining of headache, nausea, breathlessness, and swelling of his legs and body, which had ensued about four or five days previously, coincident with the onset of an attack of bronchitis to which he was subject (Fig. 297). The left heart was dilated, the liver enlarged and tender, and œdema was widespread and considerable. The blood-pressure was elevated. He was given a dietary of milk, evacuant treatment was pursued, and caffein citrate administered, and in a few days the symptoms had abated.

Coincidentally the blood-pres-

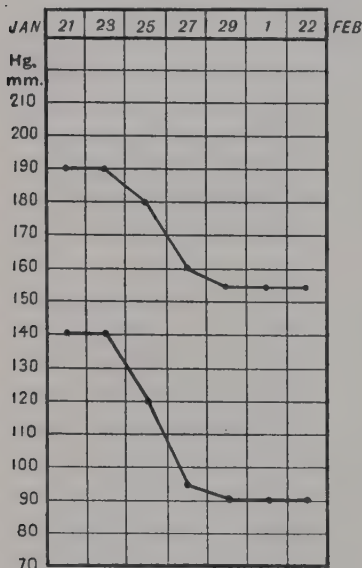


FIG. 297.—BLOOD-PRESSURE CHART.

Male, aged thirty-eight; bronchitis.

sure fell, the diastolic fall being relatively greater than the systolic. The bronchitis seems to have been responsible for an increase in the blood-pressure before which the heart gave way; with the subsidence of the bronchitis and cardiac stimulation, the pressure fell within the bounds of the cardiac strength.

Bronchitis, as is well known, imposes extra work upon the kidneys, and in nephritis even a catarrh may produce serious symptoms. A striking example occurred recently in the

wards. A man who was convalescing from nephritis contracted a catarrh which was prevalent in the ward. Coincidentally his urine, which had been copious and had contained a mere trace of blood, became scanty and of the colour of porter. The chest seemed normal, but there was a cough and a slight muco-purulent expectoration, and some reddening of the fauces.

In the baker's case (p. 534) cardiac failure occurred before an accidental elevation of the blood-pressure, but it may also fail before the permanent increase of pressure due to arterial disease, as in the case of a man, aged forty, who became breathless on the least exertion, and developed œdema of his legs whenever he was allowed out of bed, though his systolic blood-pressure was 270, and his diastolic blood-pressure 190 mm. Hg.

It is easy to lower blood-pressure. It falls notably with a sharp attack of diarrhœa, for instance, and drastic purgation and a restricted dietary can almost always reduce it considerably. But the fall is not necessarily beneficial to the patient if it results from cardiac failure. In the majority of patients, however, in the presence of symptoms, a high blood-pressure indicates imperfect elimination, and care should be taken to ensure that excretion is ample. In a large number of cases relief occurs coincidently.

A high blood-pressure in arterio-sclerosis is not necessarily an index of intoxication, for in this disease the blood-pressure is always elevated, and the "normal" pressure is that at which the heart works easily and elimination is sufficient—*i.e.*, the pressure at which symptoms are in abeyance—and it can only be gauged by repeated observations over long periods of time. One of our patients, for example, a man of sixty-one years, has had a systolic blood-pressure of between 205 and 240 mm. Hg. for the last eight years. In 1914 he had a double pneumonia, in 1915 he had symptoms of cardiac failure for a few weeks, in 1916 he had an apoplexy. But he is still going about his business, though there are now evident signs of mental and physical weakness.¹

Vasodilator drugs have been much lauded and much deprecated at different times. There is little use in giving

¹ He died in the spring of 1922.

them to a patient whose elimination is insufficient and peripheral resistance in consequence increased ; we are giving with one hand and taking away with the other ; and they have but little effect upon fibroid vessels. Their chief value is in temporary emergencies ; and their *continued* use in patients who are, so to speak, convalescent, is a confession of failure—failure to ensure that elimination is sufficient. In the presence of symptoms the blood-pressure may be temporarily elevated above the normal of the individual, and the reduction of the blood-pressure by these means may temporarily relieve distress, but it is irrational to assume that because the blood-pressure sometimes falls coincidentally with improvement, improvement will ensue whenever and however the pressure is lowered. In many cases this reduction can only be obtained at the cost of cardiac failure.

In sleeplessness and in dyspnœa vasodilators may be of service, and in angina pectoris amyl nitrite is often of value during the paroxysms, and nitroglycerine in the intervals. But the action of all drugs of this kind is very fleeting, erythrol tetranitrate, which is said to have the most prolonged action, losing its effect within five or six hours after its administration ; and we have failed to find that their continued use has had any appreciable effect upon the blood-pressure curves.

The action of the *digitalis series* (see p. 376) in cases of arteriosclerosis when cardiac failure has supervened is in the majority of cases injurious, the dyspnœa being intensified and the œdema increased. The special cardiac functions, too, may be disturbed, and an irregular pulse may give evidence of a hitherto latent heart-block, or an excessive excitability. The *pulsus alternans* may occur. Caffeine, theobromine, ammonia, alcohol, etc., are more generally useful and should be tried first. Their action should be aided by the alkaline diuretics, such as the citrate and acetate of potassium, and liquor ammonii acetatis. Strong freshly infused tea and coffee are often useful. But it must be acknowledged that in many instances these drugs prove useless, and we are forced to resort to the *digitalis* group.

French writers advocate isolated massive doses in cardiac failure, but the repeated administration of small doses seems

preferable in the cases which we are now considering, as the minor adverse symptoms can then be quickly checked by the withdrawal of the drug. A standardized tincture, the *Nativelle* granules, Trousseau's wine, or Guy's pills,¹ may be utilized; the granules and the wine scarcely ever upset the stomach. Some patients are very susceptible to mercury, and we have seen well-marked salivation occur after the administration of fourteen Guy's pills in seven days. Oral sepsis was extreme in this patient, but the use of mercury should always be carefully watched in these cases, particularly if the renal functions are also notably disturbed.

The actual height of the blood-pressure is a matter of no practical importance if elimination by the bowels is free, and we have given even large doses of *digitalis* in high-pressure cases with benefit. A woman, aged fifty-eight, was admitted to hospital orthopnoëic and very short of breath, with extensive œdema and a large effusion into the left pleural sac (Fig. 298). Paroxysmal attacks of dyspnœa occurred at night. The effusion had

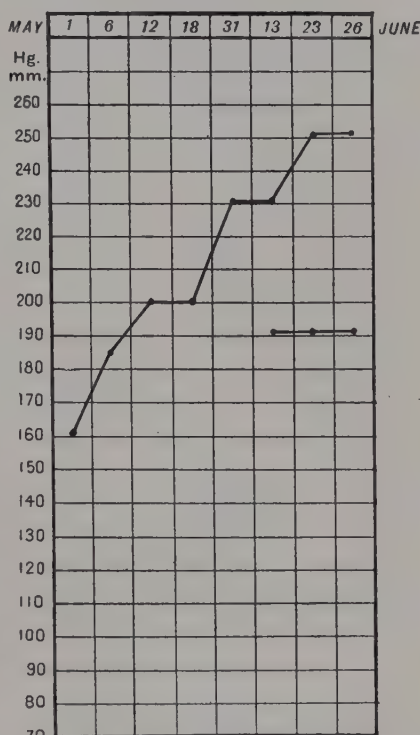


FIG. 298.—BLOOD-PRESSURE CHART.

Female, aged fifty-eight. Mitral valvular disease; cirrhotic kidneys.

to be tapped on five occasions, but she left hospital after two months' residence, fairly comfortable and without any œdema, though she was short of breath when she was walking about. Coincident with the improvement, the blood-pressure rose from 160 to 250 mm. Hg. The improvement,

¹ See p. 385.

however, was of short duration, and she returned to hospital in a moribund condition two months later.

The height of the blood-pressure is no index to cardiac sufficiency. A man, aged sixty-eight, was admitted to hospital two days after the occurrence of a right-sided hemiplegia (Fig. 299). Three days later he was more stupid than on admission, deglutition was difficult, and incontinence of urine complete, and the breathing was typically Cheyne-Stokes in character and associated with pupillary and pulse

phenomena. The extremities were cold and blue. Three days later, when improvement was distinct, the blood-pressure was practically the same, although all the evidences of venous stasis had passed away. It is difficult to realize that cardiac failure may be present with a blood-pressure of over 150 mm. Hg., but improvement ensued after the administration of strong tea and whisky and a more ample dietary—treatment which could hardly have any great effect

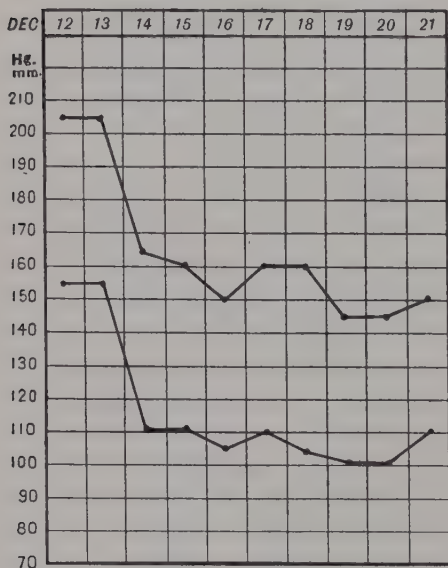


FIG. 299.—BLOOD-PRESSURE CHART.

Male, aged sixty-eight. Hemiplegia.

upon a gross cerebral lesion. In another case the demonstration was more complete. The patient, a man aged fifty-one, who was suffering from renal cirrhosis, developed cerebral symptoms which the *post-mortem* examination showed were the result of cortical emboli derived from clots in the left ventricle. His systolic blood-pressure had ranged from 170 to 210 mm. Hg.

In many of these cases of cardiac failure the whip is not required, for if some of the coals be removed from the cart,

the burden can be borne, and with adequate rest the heart regains its strength.

Cases with *symptoms of uræmic type* react well to treatment of this kind. Free purgation, packs, cupping, and an ample supply of fluid are the main elements in treatment, and the cardiac dilatation often rapidly subsides, and the pulse becomes less frequent. The administration of water is important. It can often be retained when any food is at once

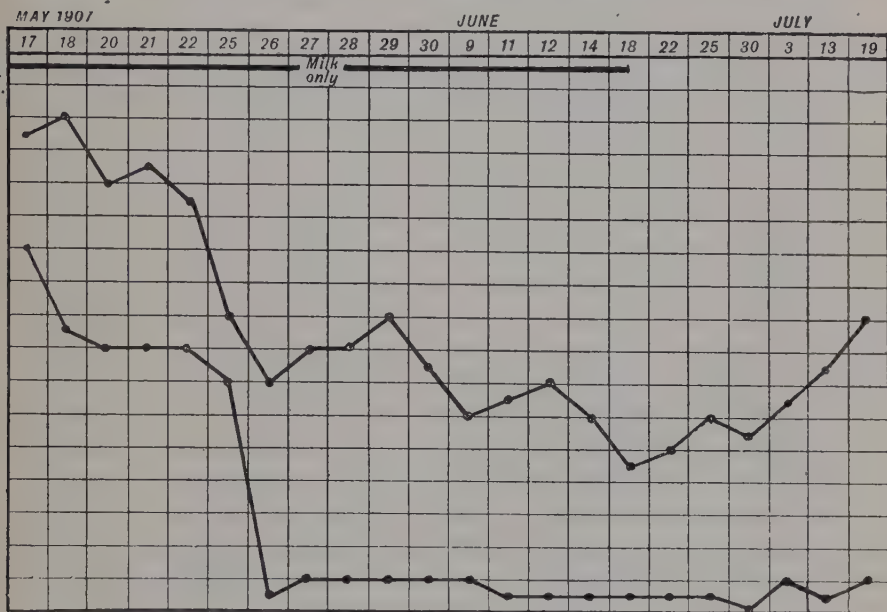


FIG. 300.—BLOOD-PRESSURE CHART.

Male, aged thirty-six; chronic nephritis.

ejected, and, of course, can be given subcutaneously or by the bowel. Continuous rectal injections, as practised by surgeons after abdominal operations, are sometimes useful. But here again the behaviour of the blood-pressure varies.

As a rule, with increased elimination and the disappearance of symptoms, the blood-pressure falls. This is well shown in Fig. 300, that of a man, aged thirty-six, who was admitted suffering from chronic nephritis, and complaining of headache, vomiting, and dimness of vision. On a diet of milk

and eliminant treatment the symptoms rapidly subsided and the blood-pressure fell, though it rose again on the resumption of a more ample dietary.

But the fall in pressure may be slight even when improvement occurs. A woman, aged forty-five, terminated her eleventh pregnancy prematurely in January. In June she

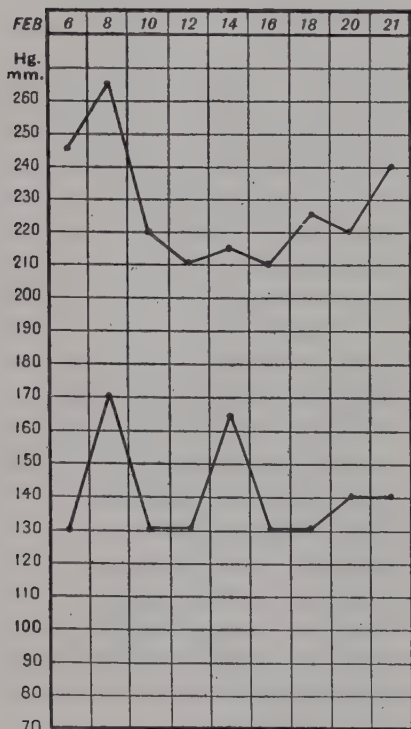


FIG. 301.—BLOOD-PRESSURE CHART.

Female, aged forty-five: chronic nephritis.

began to suffer from headache, which steadily became more severe and persistent, and at times prevented sleep. Nausea and vomiting ensued in December, and in January was very severe, almost all food being rejected at once. On admission, in February, she was extremely ill, with a pale, anxious expression, yawning continually, and complaining bitterly of headache and nausea (Fig. 301). For forty-eight hours after admission she vomited almost constantly, but the symptoms afterwards improved rapidly, and she left hospital in fair health. The blood-pressure in this case fell at first, but rose again before dismissal. Treatment was eliminant,

and she was able to retain even large quantities of water in the stomach at a time when all varieties of food were at once rejected.

The blood-pressure may even rise under similar treatment. A woman, aged forty-six, who had suffered from rheumatic fever in early life, began to be troubled, about the age of thirty-eight, with breathlessness and palpitation,

and became subject to attacks of bronchitis. Six years later œdema made its appearance, at first in the face and then in the feet, and nausea and vomiting ensued. When she was admitted to hospital a year afterwards, there was slight general œdema and a small ascitic collection, and she was emaciated and weak (Fig. 302). Rest in bed, a milk diet, and increased elimination rapidly dissipated all the symptoms, and coincidentally the blood-pressure rose from 155 to 175 mm. Hg. The only stimulant administered was some Easton's syrup. The further history is instructive.

The symptoms re-

curred, and she

was readmitted

into hospital with

similar but more

severe symptoms.

On this occasion

Trousseau's wine

was given, and

she made a slow

recovery, and

again the blood-

pressure rose from

150 to 175 mm.

Hg. She was

again admitted to

hospital, sixteen

months after dis-

missal. The

blood-pressure

was then 140 mm. Hg., and treatment failed to produce

any distinct improvement, the blood-pressure remaining

practically constant. *Post-mortem* examination revealed the

widespread nature of the disease, for there was found, be-

sides mitral stenosis and thickened degenerate vessels, well-

marked cirrhosis of the kidneys and pulmonary emphysema,

and an early cirrhosis of the liver.

In the milder forms of cardiac failure, then, eliminant

treatment is the main indication, and not infrequently is

sufficient in itself to relieve the symptoms. In more severe

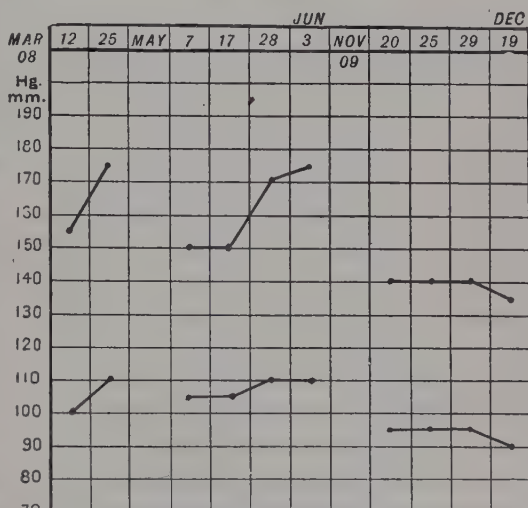


FIG. 302.—BLOOD-PRESSURE CHART.

Female, aged forty-six : mitral valvular disease ;
cirrhotic kidney.

failure cardiac stimulants may be required in addition. The essential point in treatment is, however, improved elimination ; without it stimulation is frequently unsuccessful.

The *arterial lesions* in arterio-sclerosis are often serious, and the urgency of the local lesion is apt to distract attention from the underlying cause. In aneurysm and in cardiac and renal cases the condition of the vessels is investigated as a matter of routine ; but in many other diseases, such as epistaxis, gastric ulcer, etc., in which arterial lesions are only occasionally the cause, their condition is apt to be neglected, though the bearing upon treatment is necessarily considerable.

There has been much diversity of opinion as to the relationship between aneurysm and heightened blood-pressure, a diversity which is, in part at any rate, due to erroneous deductions from the available facts. The blood-pressure is measured in the brachial artery, which is a branch of the subclavian. It is manifest that the blood-pressure in the subclavian artery cannot be high if a distensible aneurysmal sac intervenes between it and the heart ; while, on the other hand, an abdominal aneurysm will not influence the subclavian pressure. In cases of aortic incompetence, too, the aortic blood-pressure depends largely on the degree of the incompetence, and the aortic valves are often affected in cases of aneurysm. But all cases of aneurysm are combined, and a general conclusion is stated which has been based upon a collection of facts which are not truly comparable.

A similar error has been made in regard to organic mitral disease, and the pulse has been stated to be hard or soft, large or small, regular or irregular, by various writers. The frequent association of chronic renal disease and mitral stenosis has been recognized for many years in the *post-mortem* room, but the clinical application of this fact seems to have escaped notice. There are two kinds of mitral disease, as G. A. Gibson said, those with low and those with high blood-pressure, and the difference lies in the extra-cardiac lesions in the arteries and the kidneys. In the second group these systems are more or less involved ; in the first they are normal (*cf.* Figs. 289 and 290, and p. 262).

It is somewhat difficult to understand the high reputation of the iodides in this disease, for it seems certain that they exert little or no influence upon the blood-pressure, while their action upon fibrous tissue is only likely to be effective when it is of syphilitic origin. The general conclusion as to their good effects is, however, too strong to be lightly disregarded, and moderate doses may be given.¹ Care should be taken that iodism does not occur.

Mercury has a well-established reputation in arterial diseases, from its action in increasing renal and intestinal excretion, and in limiting bacterial processes in the intestines. Calomel and blue pill are the most useful preparations. The toilet of the mouth must be carefully supervised during their administration, particularly in those cases in which renal symptoms are prominent.

In patients who are more or less convalescent such drugs should be given intermittently rather than continuously, their effects being probably best marked when given every alternate week or fortnight, with occasional longer intervals of intermission.

In a general way in chronic disease all treatment must be more or less intermittent, however desirable continuous treatment may be. Few patients will bear restrictions well for many weeks on end. It is thus as a rule advisable to prescribe *courses* of treatment from time to time, rather than to lay down immutable laws as to the habits of the sufferers. We have sometimes seen the lives of elderly patients made unendurable and unhappy, without any appreciable benefit, when too rigid a *régime* had been prescribed.

¹ Iodide of potassium or sodium, hyodin, iodicin, liquor hydrargyri et arsenici iodidi. Thyroid extract is sometimes useful.

CHAPTER XXXII

ANEURYSM OF THE AORTA

Etiology.—The saying that aortic aneurysm occurs in men who have worshipped at the shrines of Venus, Vulcan, Bacchus and Mars expresses the truth, if not the whole truth, for the most important cause of aneurysm of the aorta is weakening of its middle coat by syphilis. In the vast majority of cases this is the determining cause and all other factors, such as age, sex, occupation, strain, and elevation of the aortic blood-pressure, are of subsidiary importance.

In young persons the initial weakening of the middle coat may be due, not to syphilis, but to bacterial infection of an acute nature causing mesaortitis and consequent “infective aneurysm,” as may occur in the course of malignant endocarditis, septicæmia, pneumonia and rheumatic fever. But apart from these causes, which are undoubtedly rare, syphilis is the essential cause.

The strong presumptive evidence of antecedent syphilis in most cases of aneurysm is no new discovery, for it was known to Ambroise Paré in the seventeenth, and to Lancisi and to Morgagni in the eighteenth, century. But that syphilitic aortitis is the fundamental cause of aortic aneurysm was not fully recognized until the birth of the present century. In 1895 Osler¹ wrote of alcohol, syphilis and overwork as etiological factors, but he laid no special emphasis on syphilis. Gibson,² writing in 1898, held that syphilis “probably plays a somewhat important part in the production of conditions leading to aneurysm”; but in the

¹ Osler, W., *The Principles and Practice of Medicine*, Edin. and Lond., 1895, 707.

² Gibson, G. A., *Diseases of the Heart and Aorta*, Edin. and Lond., 1898, 830.



FIG. 303.—SYPHILITIC AORTITIS WITH CHARACTERISTIC SYPHILITIC SCARRING AND ANEURYSM OF THE ASCENDING PORTION OF THE ARCH OF THE AORTA.

The aneurysmal sac is filled with a laminated thrombus. (Glasgow Royal Infirmary Museum.)

latter half of the last century the dependence of aortic aneurysm on syphilis was doubted or denied by nearly all of the most eminent authorities. Thus, as recently as 1899, Gairdner¹ was sceptical of the syphilitic origin of the disease ;

¹ Gairdner, W. T., *Allbutt's System of Medicine*, Lond., 1899, vi., 362.

but Welch,¹ Sansom² and Drummond³ emphasized its close relation to syphilis.

The recognition of the characteristic lesions of syphilitic aortitis, as distinct from atheroma, was the next link in the chain of evidence. The characteristic lesions were shown to be circumscribed plaques (Fig. 271), raised, greyish-white, and somewhat gelatinous-looking, in which spirochætes might be demonstrated. These plaques eventually form irregular, puckered, and often pigmented cicatrices in the wall of the aorta (Figs. 29, 303). The lesions, which are most often found in the ascending portion of the aortic arch, begin as a mesaortitis and cause degeneration and necrosis of the muscle and elastic fibres of the middle coat (Figs. 272, 273, 274). The affected area of the aortic wall, being thus weakened, yields to the pressure within the artery, whereby the aortic wall bulges outwards to form an aneurysm, the sac of which is formed by one or all the coats. The wall may yield over a large area, forming a fusiform aneurysm, or at a circumscribed area, forming a saccular aneurysm. In other instances the intima splits at the site of the syphilitic lesion and the sac is formed by the outer, and possibly by part of the middle coat, a "dissecting aneurysm" being thus formed.

Within the last ten years the significance of syphilis as the fundamental cause of the vast majority of aortic aneurysms has become generally recognized. It is often ten, twenty, or more, years after the primary infection that the aneurysm develops. Most of the patients yield a positive Wassermann reaction. Our own figures show that 18 of 25 cases gave a strongly positive reaction, whereas 7 were negative. No reliance can be placed on the patient's denial of syphilis. He may frankly admit having suffered from gonorrhœa or from a soft chancre, yet stoutly deny syphilis. For example, a man of thirty-seven years, suffering from an aneurysm of the ascending aorta, and yielding a strongly positive Wassermann reaction, admitted a soft sore sixteen years previously, but denied syphilis. Moreover, the fact

¹ Welch, F. H., *Med.-Chir. Trans.*, Lond., 1876, Series ii., xli., 59.

² Sansom, A. E., *Twentieth Century Practice*, Lond., 1896, iv., 492.

³ Drummond, D., *Brit. Med. Journ.*, 1908, ii., 1405.

of the patient being married and having a large healthy family does not contra-indicate syphilis. One of our cases, a woman with a family of eleven children, whose husband had died from rupture of an aneurysm, developed aortic incompetence, an aneurysm of the ascending aorta, an alternating pulse, and died of heart failure at the age of forty-three.

In all cases, and especially if the Wassermann reaction of the blood cannot be tested, careful search for other signs of syphilis may reveal scars at the base of the glans penis, periosteal nodes on the tibiæ, syphilitic leukoplakia, abolition of the knee and Achilles reflexes, the Argyll-Robertson pupil and other signs of tabes dorsalis, or of general paralysis of the insane. In our experience, however, it is rare to find any unequivocal clinical signs of active syphilis in patients affected with aortic aneurysm, and a positive Wassermann reaction is often the only definite clinical proof of luetic infection ten, twenty, or thirty years previously. A single negative reaction does not contra-indicate syphilis. We have obtained negative reactions in patients who were proved by *post-mortem* examination to have been suffering from syphilitic aortitis; moreover the reaction may become positive after a provocative dose of salvarsan or one of the other organic arsenical preparations.

On *post-mortem* examination the characteristic lesions of syphilitic aortitis (Figs. 29 and 271) are almost invariably found, but visceral gummata are comparatively rare.

Age and Sex.—Aortic aneurysm is most frequent between the ages of thirty and forty-five, and occurs more often in males than in females, the ratio for the two sexes, as given by different writers, varying from 4:1 to about 8:1. The statistics of the Registrar-General for Scotland show that the number of deaths due to aneurysm in seven years (1911 to 1917) was 982, of which 758 were in males, 224 in females. In England and Wales during 1917 there were 995 deaths due to aneurysm, 770 being in males, 225 in females. On the basis of these figures the ratio of males to females is 3·4:1. The age incidence of death from aneurysm is shown in the following table, compiled from the Reports of the Registrar-General for Scotland, for the years 1911 to 1917:—

	All ages	-1	1-	5-	10-	15-	25-	35-	45-	55-	65-	75 and over
Males ..	758	—	—	—	1	3	24	176	218	202	100	34
Females	224	1	—	—	2	3	7	18	54	59	47	33
Both sexes ..	982	1	—	—	3	6	31	194	272	261	147	67

In this table the site of the aneurysm is not indicated, but the deaths occurring below the age of twenty-five were probably not due to aneurysm of the aorta. In early life death by rupture of an aneurysm of a cerebral artery is not so rare. For example, a boy aged sixteen, having been found comatose in a public lavatory, died within two hours after admission to our wards. An aneurysm, rather larger than a pea, of the left middle cerebral artery had ruptured. The viscera, and the arteries apart from the aneurysm, appeared to be healthy.

Aortic aneurysm is most frequently fatal, in men, between the ages of fifty and fifty-four, and in women, between sixty and sixty-four. In women fusiform aneurysm of the aorta is rare. In 1917 the crude death rate among the civilian population of England and Wales, from aneurysm, at all ages was, for males 51 per million living, for females 11 per million living, and for all persons 28 per million. The age and sex incidence in our own series of aortic aneurysms is shown in the following table, which records the age at which the patients came under observation, not their age at death.

	All ages	30-	35-	40-	45-	50-	55-	60-	65 and over
Males	37	2	4	7	5	9	4	5	1
Females	4	—	—	2	—	1	—	—	1
Both sexes ..	41	2	4	9	5	10	4	5	2

In Drummond's series of 300 cases, the age in 128 cases of saccular aneurysm was under forty-five, while 72 were between forty and forty-five. Fusiform aneurysm usually occurs a little later, two-thirds of the cases being between the ages of forty-five and fifty-five.

Occupation.—Aortic aneurysm is most frequent in iron-

moulders and steel-workers, in dock labourers and brewers' draymen, and in those who are engaged in other forms of arduous manual labour involving prolonged stress and often sudden severe strain. It is because physical strain and stress raise the intra-aortic blood-pressure that men engaged in hard manual labour are more prone to develop aortic aneurysm than those whose occupation is more sedentary. Moreover the casual labourer is more addicted to alcoholic excess, and is therefore more liable to acquire syphilis, than his more highly educated, but perhaps poorer, neighbour. In former years aortic aneurysm was common in soldiers and especially in gunners, but with the efficient diagnosis and treatment of syphilis now prevailing in the Army, it may be expected that aortic aneurysm will become comparatively rare in soldiers. Aneurysm is not frequent in women because they are relatively seldom infected with the *Spirochæta pallida*, and because their occupation does not usually involve severe physical stress.

Morbid Anatomy.—Aneurysms of the aorta are (1) *Saccular*, by the development of a localized pouching or bulging outwards of the aortic wall, (2) *Fusiform*, with uniform dilatation of the lumen of the aorta over a certain area, and (3) *Dissecting*, in which the inner and middle, and possibly even the outer, coats have ruptured, and the blood has passed out of the lumen of the artery into adjacent tissues which form the sac of the aneurysm.

Site.—The arch of the aorta, and especially its ascending portion, is the most frequent site of aneurysm, because this is the predominant site of syphilitic aortitis, and also because it is the ascending portion of the arch upon which falls the maximal strain of increased pressure each time the blood is expelled from the left ventricle. According to Drummond the site of saccular aneurysm of the thoracic aorta is : Ascending aorta, 73 ; transverse, 76 ; descending, 30. The figures tabulated by Osler were : Ascending aorta, 159 ; transverse, 113 ; descending, 49 ; abdominal aorta, 83 cases. In Drummond's series of thoracic aneurysm, 179 were saccular, 39 fusiform, and 7 dissecting.

Size.—Aortic aneurysms vary in size from that of a cherry

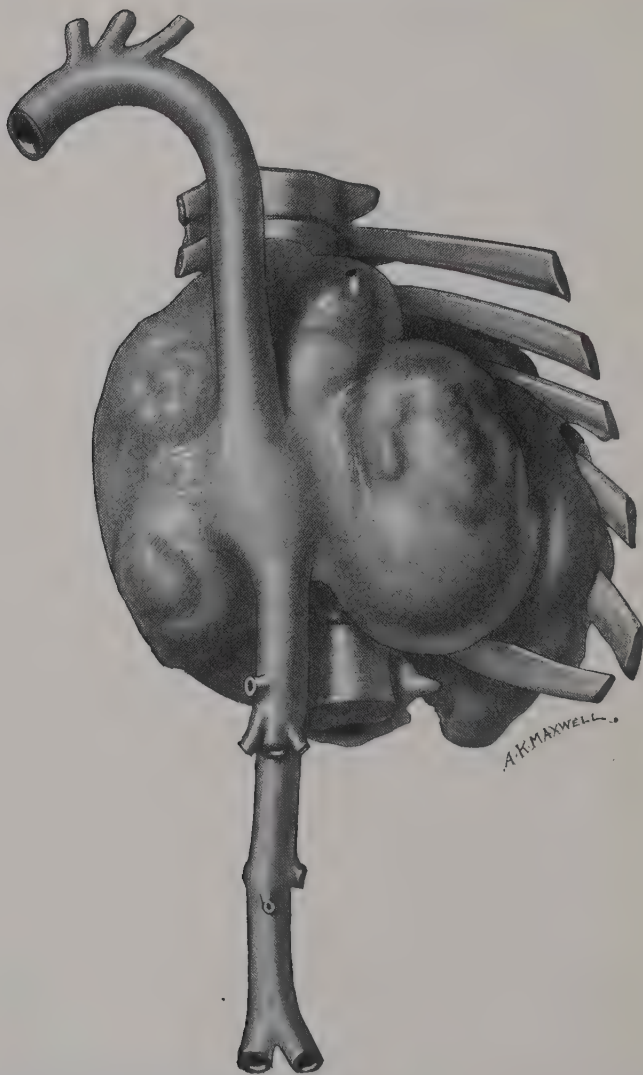


FIG. 304.—LARGE SACCULAR ANEURYSM OF THE DESCENDING THORACIC AORTA, ERODING THE BODIES OF FIVE VERTEBRÆ AND RIBS.

(From a specimen in the Museum of the Royal College of Surgeons of Edinburgh.)

stone to that of a man's head. In form, aneurysms differ greatly, but a saccular aneurysm is usually more or less globu-

lar, though there may be secondary sacs arising from the original, main, aneurysm. In some cases there is more than one aneurysm. In a dock-labourer aged thirty-nine, in whom the ascending portion of the aortic arch was dilated, measuring 17 cm. in circumference, one aneurysmal sac the size of a walnut arose from the left posterior aortic sinus, and a second sac, as large as half a Tangerine orange, arose from the transverse aorta. The opening from the aorta into the sac may be small, admitting only the tip of the little finger, or may be large. The sac is more or less filled with laminated thrombus (Fig. 303). The sac of a large aneurysm presents no trace of intima or of media, but is composed of fibrous tissue which is derived partly from the adventitia. On the inner wall of the sac there are often calcareous plates, and dense fibrous adhesions are formed between the sac and the structures which are displaced by its growth. As the aneurysm enlarges still further and erodes the adjacent tissues by pressure, the sac may be formed in part by the sternum, the vertebræ or ribs (Fig. 304), by lung, trachea (Figs. 317, 319), skin, or other structure which is being eroded.

Other portions of the aortic arch usually present cicatrices characteristic of old-standing syphilitic aortitis; and in some instances the aortic wall presents calcareous plaques and other signs of atheroma. An aneurysm of the ascending portion of the aortic arch is often combined with incompetence of the aortic valve, likewise of syphilitic origin.

SYMPTOMS AND SIGNS OF ANEURYSM OF THE AORTA

I. ANEURYSM OF THE ARCH OF THE AORTA

The symptoms in individual cases vary in large measure according to the site and size of the aneurysm. In some individuals, and particularly in those having a small aneurysm at, or immediately above, the aortic sinuses, symptoms and signs are often entirely latent. The case is regarded as one of aortic incompetence, and the presence of an aneurysm may not be suspected until an autopsy reveals it. In these cases death is often sudden from rupture of the aneurysm into the pericardium. Even an aneurysm of large size, compressing adjacent structures and eroding bone, may be unattended by

any symptoms. Other patients have severe and distressing symptoms although abnormal physical signs, apart from radiographic evidence, are lacking. Broadbent's classification of aneurysms into those with symptoms and those with signs is a useful one, but it is necessary to bear in mind that an aortic aneurysm, and particularly one of the descending thoracic aorta, may produce neither symptoms nor signs, and be recognizable solely by radiographic examination. The vast majority of cases, however, present both symptoms and signs. Of the former, pain, throbbing and dyspnoea are the most frequent. It is convenient to consider the symptomatology in relation to the various systems.

I. Circulatory System.—*Pain* is undoubtedly the most frequent, and in the vast majority of cases the earliest, symptom. It is usually constant, varying, however, in severity from time to time, being exaggerated by physical exertion, and tending to lessen when the patient is resting. The site of the pain differs according to the situation of the aneurysm, and to the direction of its growth, and consequently according to the structures which it is compressing or eroding. The severity of the pain depends largely upon the size of the aneurysm, the rapidity of its growth, the compression of nerves, and the erosion of bone. In many cases the pain is anginal in character. Pain may be referred to the præcordia, but more often to the upper part of the sternum, and particularly to the manubrium, than to the apical region, and more often to the right than to the left side of the chest. One of our patients had suffered from pain, which was considered to be "neuralgia of the heart," for two years before he died of an aneurysm of the aortic arch. Like cardiac angina, the pain may radiate from the front of the chest into the neck or into the arm. More characteristically, however, the pain of aortic aneurysm is constant and of a gnawing character; it may be predominantly nocturnal, and is only partially relieved by morphia. One of our patients used to obtain relief from the nocturnal pain by performing dumb-bell exercises until he sweated.

An aneurysm of, or immediately above, the aortic sinuses seldom causes pain. That of an aneurysm arising a little

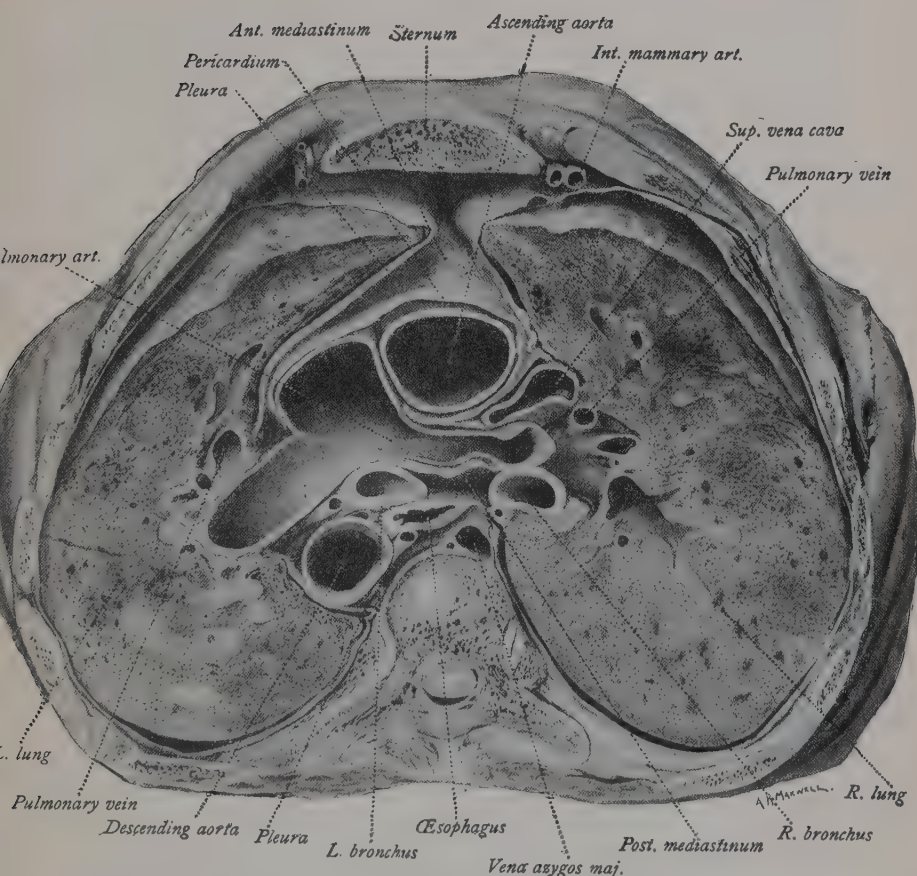


FIG. 305.—TRANSVERSE SECTION OF THORAX AT LEVEL OF THE UPPER BORDER OF THE SEVENTH DORSAL VERTEBRA POSTERIORLY, AND AT THE LEVEL OF THE SECOND RIB ANTERIORLY, TO SHOW THE RELATION OF THE ASCENDING AND DESCENDING AORTA TO OTHER STRUCTURES.

(From the Museum of the Royal College of Surgeons of England.)

higher up on the ascending portion of the arch is usually referred to the præcordia or to the middle of the sternum, and often radiates to the right shoulder, down the right arm, or into the back between the shoulders. The pain of an aneurysm of the transverse aorta, which is growing forwards, is usually located in the upper part of the sternum, whereas if the aneurysm is extending backwards the pain is chiefly in the interscapular

region. In cases of aneurysm of the descending portion of the arch, the pain is referred mainly to the left scapula or shoulder, down the left arm, especially if the left brachial plexus is compressed, or may radiate around the chest owing to compression of the intercostal nerves or nerve roots.

The distribution of the pain, however, does not always conform to that which might be expected. Thus in one of our cases a large aneurysm of the descending thoracic aorta, extending into the right side of the chest, caused pain solely in the right infra-mammary region. Partial relief from the constant, agonizing pain may be afforded by change of posture. Thus a patient suffering from an aneurysm of the ascending portion of the arch may find some relief by lying in the prone position. Hyperalgesia can often be elicited over the area to which the pain is referred.

Throbbing.—The consciousness of the aneurysmal pulsation may be the outstanding symptom. This is usually referred to the thorax, but may be experienced in the supra-sternal fossa or in the side of the neck, especially if the innominate artery, as well as the aortic arch, is the seat of an aneurysm.

Although the facial expression is often that of pain and anxiety, there is no undue pallor unless the aneurysm is associated with anæmia or with aortic incompetence of severe degree. Nor is the face cyanosed unless the aneurysm is compressing the respiratory passages or the superior vena cava. If this vessel is compressed, the upper part of the trunk, the face and the upper limbs become livid, while the veins of the neck and the superficial veins of the upper part of the chest are distended. Venous engorgement may be the chief condition of which the patient complains. Thus, a man aged thirty-four, who admitted syphilis and alcoholism, and who had slight dyspnœa but no pain, complained of great swelling of the cervical veins for six weeks before a large aneurysm of the transverse aorta ruptured into the pericardium. Engorgement of the veins, or œdema, of one arm and hand indicates compression of the innominate vein or of one of the subclavian veins.

Inspection of the chest may reveal a large pulsatile and expansile swelling in any part of the chest, but most frequently to the right of the sternum above the level of the third rib.

But an aneurysm has attained a large size before it protrudes on the chest wall. More often there is visible only a slight, diffuse impulse of the upper part of the præcordia, of the manubrium, or of the chest on either side of this bone. To observe the slighter degrees of pulsation, the patient should be seated between the source of light and the observer, and the eye should be on a level with the patient's chest. The apex impulse of the heart is seldom displaced downwards and outwards, unless the heart be enlarged from co-existent valvular disease or from some other cause such as chronic interstitial nephritis. The apex impulse may, however, be at an abnormal site if the heart itself is displaced by the aneurysm.

Palpation.—In all except the largest aneurysms, palpation yields more definite information than inspection. Every part of the chest wall should be palpated systematically, with the palm of the hand and the fingers firmly applied to the thorax. It is above the level of the third rib that the slight diffuse heaving impulse, systolic in time, so characteristic of aneurysm, is usually felt. In many cases aneurysmal pulsation is felt more distinctly if the chest is firmly compressed between the two hands, one on the front, the other on the back of the chest. This method of palpation is a routine part of our examination in every doubtful case. The pulsation of a substernal aneurysm is sometimes very pronounced and, as was emphasized by Balfour,¹ it may be even more forcible than that of the heart.

The palpable impulse of an aneurysm, when timed by the apex impulse, appears to be practically synchronous with the latter. A tracing, however, will prove that the aneurysmal impulse begins after the apex impulse, and before the pulse wave reaches the carotid, brachial, or radial artery (Fig. 306).

The forcible closure of the aortic valve may also be felt, and more rarely a thrill, which is often most pronounced in the episternal notch or over the manubrium sterni. It is in cases of arterio-venous aneurysm, with a free communication between artery and vein, that a thrill is most obvious. In the episternal notch aneurysmal pulsation can often be felt well, but a systolic impulse in this region is not necessarily

¹ Balfour, G. W., *Clinical Lectures on Diseases of the Heart and Aorta*, Lond., 1898, 407.

that of an aneurysm, whether saccular or fusiform, for the same degree of impulse may be felt in cases of aortic incompetence, and also in exophthalmic goitre and other forms of throbbing, but not dilated, aorta.

Percussion.—The heart is sometimes found to be enlarged, but more frequently it is displaced to the right or left by the growth of the aneurysm. Percussion may reveal a definite area of impaired resonance, or even of absolute dullness, beyond and usually above the cardiac dullness, and correspond-

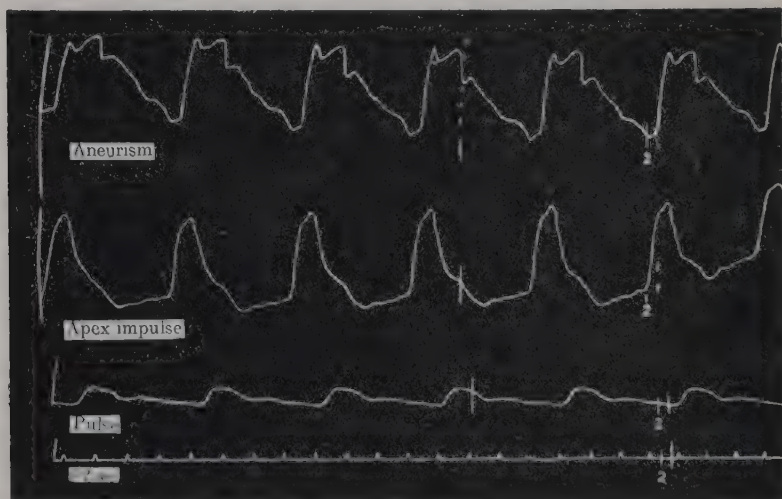


FIG. 306.—SIMULTANEOUS TRACINGS FROM AN AORTIC ANEURYSM PULSATING AT THE RIGHT NIPPLE, THE APEX IMPULSE, AND ARTERIAL PULSE.

The aneurysmal pulsation follows the apex impulse (2) and precedes the arterial pulse.

ing approximately in form and in size with the area of extra-cardiac pulsation (Fig. 307). The area of dullness yielded by an aneurysm of the aorta is seldom, if ever, a safe indication of the size of the aneurysm because only a portion of the latter is in contact with, or near to, the thoracic wall; and the aneurysm is usually much larger when seen by radioscopy, or at the autopsy, than would have been suspected from the area of dullness.

If percussion is to be of value in the recognition of aortic aneurysm in its early phases, the note at corresponding points

on the two sides, front and back, must be compared carefully, because the degree of dullness caused by an aneurysm is slight unless it is growing forwards; but an area of dullness may be detected in the left interscapular region alone (Fig. 309). The difficulty of determining an aortic aneurysm by percussion is greatest in a stout, deep-chested man with a deep-seated aneurysm.

Auscultation is less serviceable than palpation or percussion. Over the area of dullness the second sound is usually heard well, indeed it is often loudly accentuated and of ringing

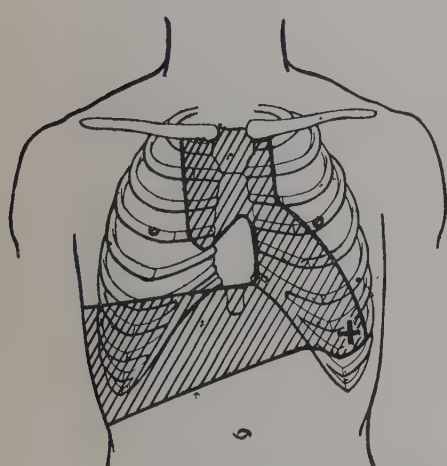


FIG. 307.—DIAGRAM SHOWING THE AREA OF DULLNESS IN A CASE OF ANEURYSM OF THE ASCENDING AND TRANSVERSE AORTA.

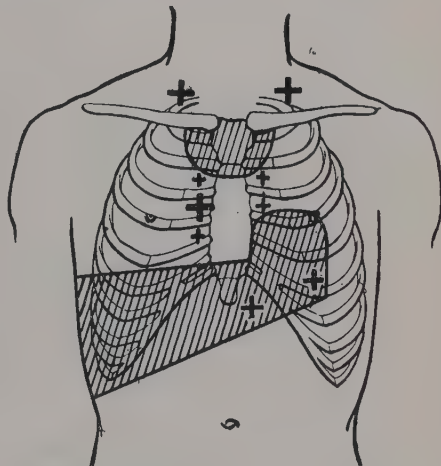


FIG. 308.—DIAGRAM SHOWING AREA OF DULLNESS IN A CASE OF ANEURYSM OF THE ARCH OF THE AORTA AND AORTIC VALVULAR DISEASE; AND THE DISTRIBUTION OF THE VISIBLE PULSATION (+).

character. A systolic murmur is much less common. It may be due to eddies in the sac, or to compression of the aorta by a saccular aneurysm, but is generally a systolic murmur transmitted through the aneurysm from a sclerosed aortic valve. Unless there be aortic incompetence, a diastolic murmur is never heard over an aortic aneurysm. A few isolated cases have been recorded of a diastolic murmur audible over an aneurysm, and yet the aortic valve was subsequently proved to be competent;¹ but in these cases the sub-

¹ Finlayson, J., *Glasgow Med. Journ.*, 1896, xlv., 299.

aortic muscle sphincter, which in health aids the aortic valve in closing the orifice, was doubtless at fault.¹

The Arteries.—The walls of the brachial, radial, and other accessible arteries are not necessarily thickened, indeed they are often remarkably soft in view of the man's age and occupation. The pulses in both radial arteries are usually equal in volume and synchronous. It is only when the innominate, or the right or left subclavian arteries are involved or compressed by the aneurysm, or when there is co-existent end-

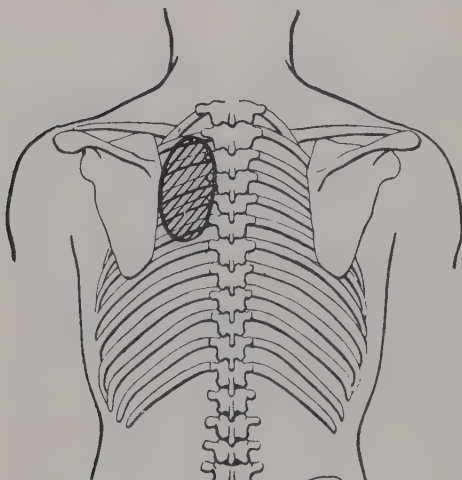


FIG. 309.—DIAGRAM SHOWING AREA OF DULLNESS IN A CASE OF ANEURYSM OF THE DESCENDING AORTA, THE SIZE OF A COCONUT, WHICH PROJECTED BACKWARDS AND ERODED THE RIBS AND THE VERTEBRAL COLUMN.

arteritis, with resultant narrowing of the lumen of one of those arteries, that the volume of the brachial and radial pulses becomes unequal on the two sides. Even although the pulse at one wrist is decidedly smaller than that in the other, they are practically synchronous. A slight retardation of the smaller radial pulse, amounting at most to a minute fraction of a second, may be demonstrated in

exceptional cases by means of graphic methods, but the retardation is never perceptible to the finger. Statements to the contrary are based on a misapprehension. Care must be taken to exclude the error arising from an abnormal distribution of one radial artery. We have seen more than one case in which failure to compare the brachial as well as the radial pulses led to an erroneous diagnosis of aortic aneurysm.

Radiographic Examination.—The radiographic signs of thoracic aneurysm are usually distinct. No other method

¹ Stewart, H. A., *Johns Hopkins Hospl. Bull.*, 1909, xx., 209.

of examination furnishes information so clear and precise regarding the state of the thoracic aorta in its whole course. The patient should preferably be examined in the erect attitude. We begin by screening him with sagittal, dorso-ventral illumination, the patient facing the screen as in Fig. 310, A, and we observe the form, size and pulsations of the cardiac

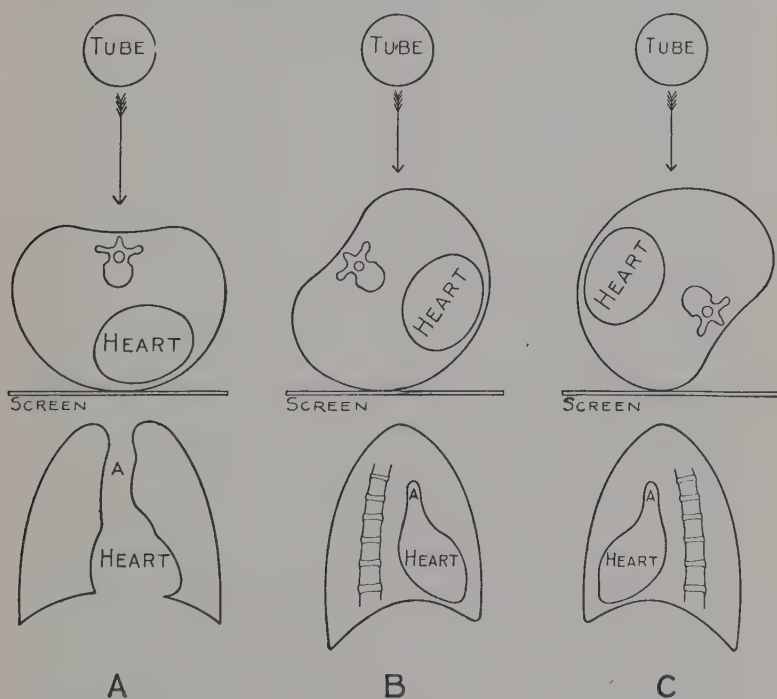


FIG. 310.—DIAGRAMS SHOWING THE RADIOGRAPHIC FORM OF THE NORMAL HEART AND AORTA (A).

A, Sagittal dorso-ventral illumination.

B, Left oblique position (rotation 45° to left).

C, Oblique position (rotation 225° to left).

and aortic shadows. The patient should then rotate slowly toward his left until, having rotated through the entire circle, he is again facing the screen. The form of the aortic shadow should be studied at all degrees of rotation. Oblique illumination is often the most instructive, especially when the patient has rotated (1) 45° to the left, bringing his right shoulder

towards the screen, his left shoulder towards the tube (Fig. 310, B), and (2) 225° to the left, when the left shoulder is towards the screen, the right shoulder towards the tube, as in Fig. 310, C.



FIG. 311.—RADIOGRAM. SACCULAR ANEURYSM OF ASCENDING AND TRANSVERSE PORTIONS OF THE ARCH OF THE AORTA. ($\times \frac{2}{3}$) (J.R.R.)

The form, size and density of the aortic shadow undergo alteration from the normal at the site of the aneurysm. On sagittal illumination, whether dorso-ventral or ventro-dorsal, the aortic shadow is seen to be broadened and to project unduly

to the right or left of the sternum (Figs. 311, 312, 313). Oblique illumination reveals, instead of the normal conical aortic shadow, one that is more or less globular and which encroaches on the retro-sternal, or retro-cardiac, space, or on both. An

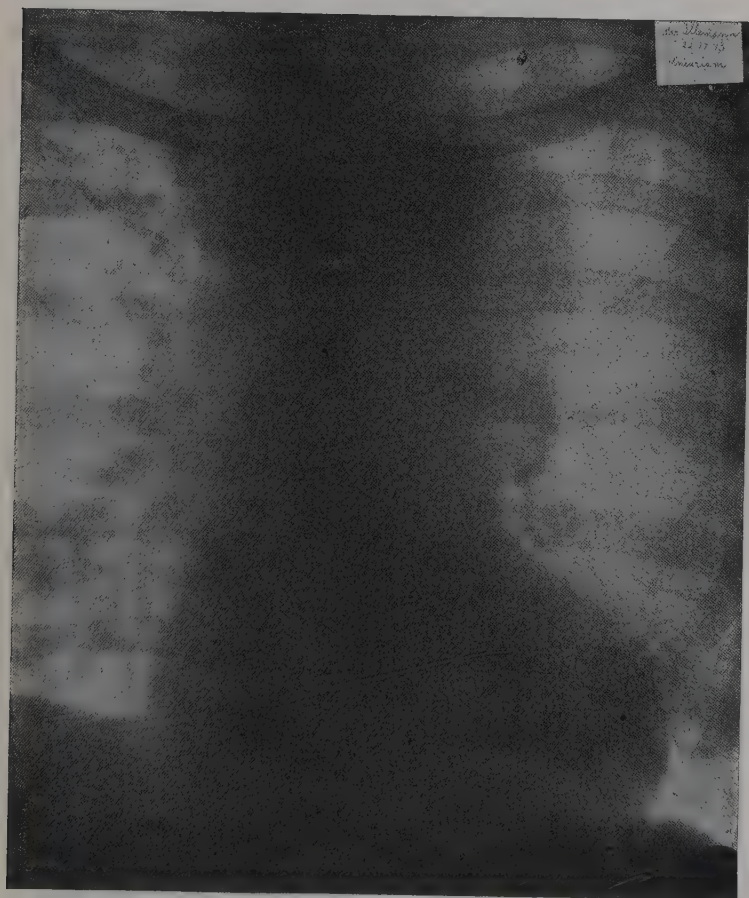


FIG. 312.—RADIOGRAM. SACCULAR ANEURYSM OF TRANSVERSE PORTION OF THE AORTIC ARCH. HYPERTROPHY OF THE LEFT VENTRICLE. ($\times \frac{2}{3}$.) (J. R. R.)

aneurysm of the ascending portion of the arch, seen by sagittal illumination, usually projects towards the right side of the sternum and encroaches on the right lung (Fig. 311). Oblique illumination may show that the sac also projects forwards,

encroaching on the upper part of the retro-sternal space. An aneurysm of the transverse aorta is often best demon-



FIG. 313.—RADIOGRAM. ANEURYSM OF THE TRANSVERSE PORTION OF THE AORTIC ARCH AND OF THE DESCENDING THORACIC AORTA. THE LEFT RECURRENT LARYNGEAL NERVE WAS PARALYSED. ($\times \frac{2}{3}$.)

strated by oblique illumination, being seen to occupy part of, or to fill, the retro-sternal or retro-cardiac space. When

examined sagittally an aneurysm of the descending portion of the arch, or of the descending thoracic aorta, casts a shadow immediately to the left of, and above, that of the heart (Fig. 313), but an aneurysm in these situations is better demonstrated by oblique illumination. The upper part of the retro-cardiac space should be studied carefully, for it is there that an

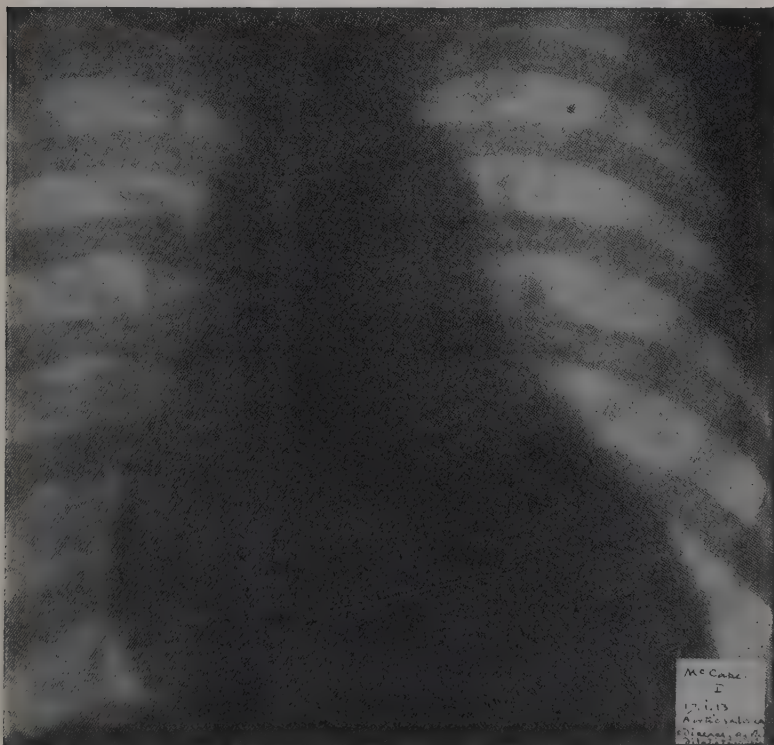


FIG. 314.—RADIOGRAM. AORTIC INCOMPETENCE AND DILATATION OF THE AORTIC ARCH. ($\times \frac{3}{2}$). (J. R. R.).

aneurysm which was not visible on sagittal illumination may be detected.

An aneurysmal shadow is usually dense, even if it be small, and its obvious pulsation is well seen on the screen. Radiograms are not without value, but for diagnostic purposes radioscopy is always preferable.

The condition which is most likely to be mistaken for an

aortic aneurysm is simple dilatation of the aorta, and particularly of its ascending portion, a condition which is frequently seen as a sequel to incompetence of the aortic valve. Fig. 314 shows the broadening of the aorta, together with the cardiac hypertrophy of this valvular lesion. In other cases, although there is no saccular aneurysm, we find a more general broadening and increased opacity of the aortic shadow, indicating a fusiform aneurysm of the aorta. Minor degrees of

aortic dilatation, causing slight broadening and increased density of the aortic shadow, in comparatively young men are

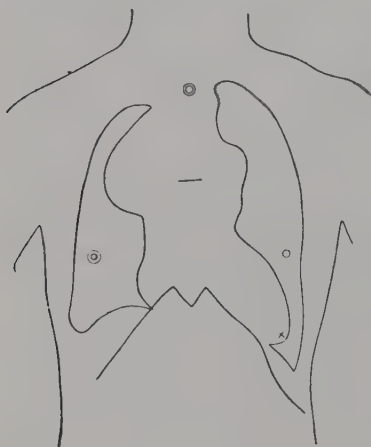


FIG. 315.—ORTHODIAGRAM. ANEURYSM OF ASCENDING AND TRANSVERSE PORTIONS OF THE AORTIC ARCH.

Male, aged sixty-eight.

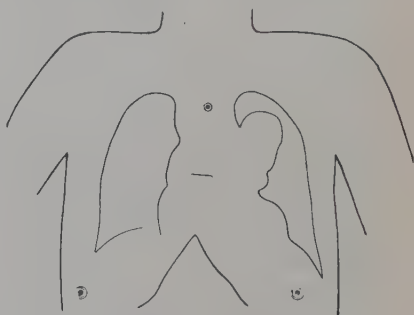


FIG. 316.—ORTHODIAGRAM. ANEURYSM OF THE DESCENDING PORTION OF THE AORTIC ARCH AND OF THE DESCENDING THORACIC AORTA.

always suggestive of, but not necessarily due to, syphilitic aortitis.

II. Respiratory System.—*Dyspnœa* is commonly caused by large aneurysms compressing the bronchi and causing collapse of part of one lung, more rarely of the whole of one lung. In these cases the dyspnœa is more or less constant. The dyspnœa which is due to compression of the trachea by an aneurysm of the transverse portion of the arch develops at a comparatively early stage, is usually severe, and is attended by noisy breathing (tracheal *stridor*). This may be either constant or paroxysmal, and when well marked has been

designated the "leopard growl" (Wyllie).¹ Tracheal compression may result from a comparatively small aneurysm; in one of our cases it was no bigger than a mandarin orange.

Dyspnœa is sometimes paroxysmal, possibly because of

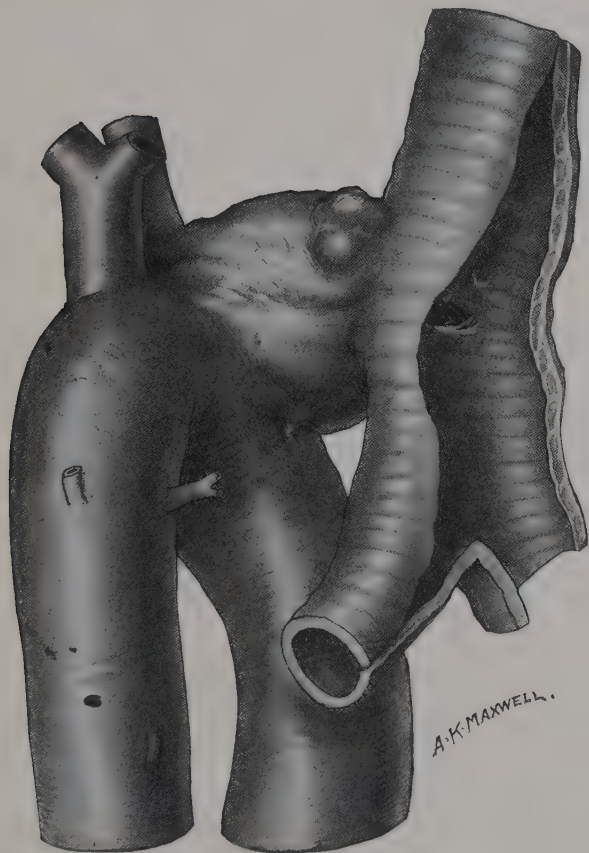


FIG. 317.—SACULAR ANEURYSM OF TRANSVERSE PORTION OF AORTIC ARCH, ADHERENT TO AND RUPTURING INTO THE TRACHEA.

(From a specimen in the Museum of the Royal College of Surgeons of Edinburgh.)

intermittent vagal compression, and may simulate bronchial asthma. One man, forty years of age, had been taking stramonium, belladonna and adrenalin without benefit for several

¹ Wyllie, J., *Edin. Hosp. Rep.*, 1893, i., 48.

months before he was admitted to hospital, where he was found to have a large aneurysm of the transverse portion of the arch, compressing the left subclavian artery, causing complete paralysis of the left recurrent laryngeal nerve and marked stridor.

Cough is usually a later symptom than pain. Thus, an iron-moulder, aged forty-four, having a small aneurysm of the transverse aorta, suffered for five months from pain in the left side of the chest, which felt as if gripped in a vice, before he began to complain of cough. The cough is sometimes of peculiar character, lacking its initial sudden explosive quality, and therefore has aptly been termed by Wyllie the bovine cough. This arises in cases of recurrent laryngeal paralysis (*see* p. 567). In contrast to the bovine cough is the harsh, strident cough with a ringing or brassy character, designated by Wyllie the gander cough, in cases with direct compression of the trachea. Coughing often induces, or greatly aggravates, the pain.

Hæmoptysis resulting from congestion or erosion of the mucous membrane of the trachea, or of one of the large bronchi, may occur in the terminal phases of the patient's illness; but after the aneurysm first begins to "weep" in this manner he may survive for several weeks, or months, before the sac ruptures into the trachea or bronchus.

Physical Signs referable to the Lungs.—Apart from the actual protrusion of an aneurysm upon the chest wall, there may be alterations in the form of the chest owing to compression of a main bronchus, or of one of its larger branches, and collapse of the corresponding portion of the lung. The bronchial occlusion leads to retention of secretion in the bronchi and to bronchiectasis, with expectoration at times of abundant muco-purulent sputum.

Careful comparison of the air-entry into the two sides of the chest should form part of the routine examination in every case in which aortic aneurysm is suspected. Compression, with consequent occlusion, of a bronchus produces defective expansion or even retraction of part of the chest wall, impairment of the percussion note, and enfeeblement of the breath sounds, of the vocal fremitus and of the vocal resonance. The area over which these alterations are manifest

varies according as it is a main bronchus, or one of its branches, which is occluded. In other cases the whole of one lung, or one of its lobes, usually the upper lobe, presents the physical signs indicating consolidation ; and at the autopsy the affected part is found in a state of grey hepatization.

Tracheal tugging (Oliver's sign) may be observed if an aneurysm has become adherent to the trachea or to the left bronchus. To elicit this sign, the patient should bend the head backwards ; the two forefingers are then pressed below the cricoid cartilage in order to pull it upwards. A downward tug of the cricoid and trachea may then be felt, synchronous with each ventricular systole (Fig. 318). The sign is very suggestive of an aneurysm of the transverse aorta, but is not pathognomonic. In our experience tracheal tugging is a

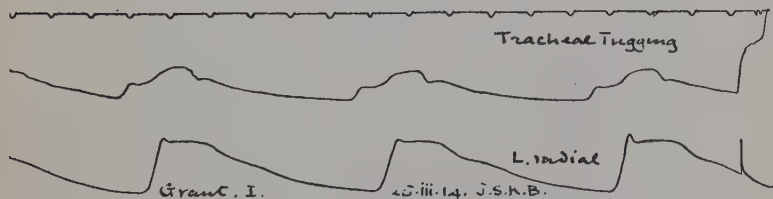


FIG. 318.—THE UPPER CURVE SHOWS THE DOWNWARD MOVEMENT OF THE CRICOID CARTILAGE. AS THE TAMBOUR WAS DIRECTED UPWARDS, THE RECORDING LEVER MOVES UPWARDS WITH THE DOWNWARD MOVEMENT OF THE PART. ANEURYSM OF TRANSVERSE AND DESCENDING AORTA.

rare sign. A common error is that of mistaking the carotid pulsation for tracheal tugging. Fixation of the larynx during deglutition may be observed if the aneurysm is adherent to the trachea or left bronchus.

III. Nervous System.—The three principal manifestations referable to the nervous system are pain (*see* p. 552), laryngeal paralysis, and irritation or paralysis of the sympathetic cord.

Laryngeal Paralysis is usually unilateral, and is more common on the left than on the right side, because it is the left recurrent laryngeal nerve, winding around the transverse portion of the aortic arch, which is most frequently subjected to compression. The right recurrent laryngeal nerve winds around the right subclavian artery and lies almost wholly above the clavicle.

The abductors of the vocal cord, as was shown by Semon, are the first to be paralysed. The adductors are still unaffected, the voice is normal, the cord lies in the middle line, and the abductor paralysis is detected only by laryngoscopic examina-

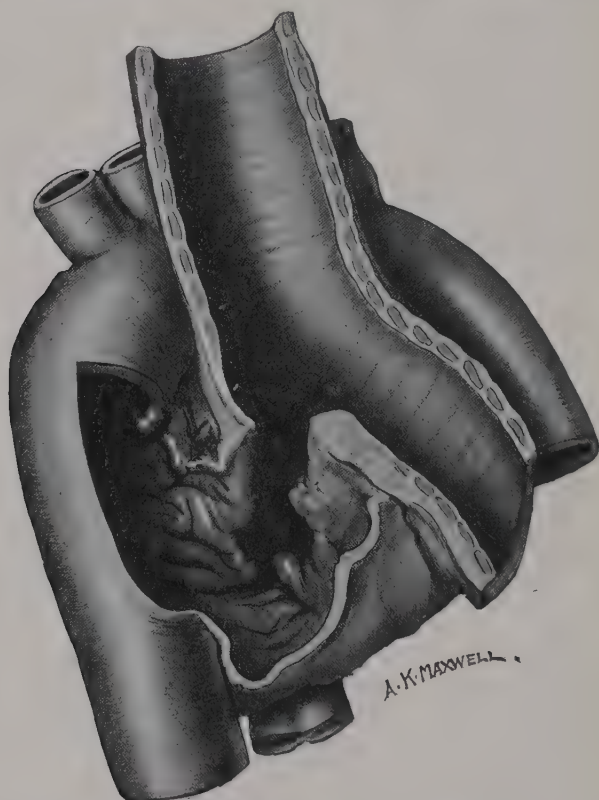


FIG. 319.—ANEURYSM OF THE DESCENDING AORTA NEAR ITS COMMENCEMENT. RUPTURE INTO THE LEFT BRONCHUS.

From the body of one of Buonaparte's Imperial Guard. While, in joy at Buonaparte's return from Elba, he was crying "Vive l'Empereur!" the aneurysm burst into the trachea. (From a specimen in the Museum of the Royal College of Surgeons of Edinburgh.)

tion which reveals failure of the cord to move outwards during full inspiration. At this stage, bilateral spasm of the adductors, causing violent paroxysms of dyspnoea, arises in some instances. As the compression of the nerve increases,

the adductors of the cord become paralysed also. The cord then lies in the cadaveric position, the voice is husky and hoarse, and the cough acquires the bovine character (*see* p. 566). In some instances the voice cannot be raised above a whisper.

Paralysis of the left recurrent laryngeal nerve is more frequently due to aortic aneurysm than to tumour. The right recurrent laryngeal nerve may be compressed by an aneurysm of the right subclavian artery, or by pleural adhesions at the apex of the right lung, with resultant paralysis of the right vocal cord. Paralysis of either nerve may be due to a nuclear lesion (e.g. bulbar paralysis or tabes dorsalis), or to compression or neuritis of the vagus trunk. In recurrent laryngeal paralysis the larynx is not anæsthetic, whereas it is so if the lesion be situated above the origin of the superior laryngeal nerve.

Compression of the Sympathetic Cord.—In the early stage irritation of the sympathetic cord causes dilatation of the pupil, increased width of the palpebral fissure and sometimes exophthalmos, on the affected side. Later, the pupil is contracted because of sympathetic paralysis and there is some enophthalmos with narrowing of the palpebral fissure. The left eye is more often affected than the right. Permanent and considerable inequality of the pupils is a most valuable diagnostic sign.

Inequality in size of the pupils may, however, be due to tabes dorsalis, to lesions affecting the third cranial nerve, or to antecedent iritis. In the latter case the affected pupil is not only small but also irregular in outline and fixed. In one woman, a small irregular immobile left pupil and an aberrant distribution of the left radial artery led to an erroneous diagnosis of aortic aneurysm. If tabes dorsalis, paralysis of the third nerve, and iritis are excluded, inequality in size of the pupils is almost certainly of intrathoracic origin, and is due either to a mediastinal tumour or to the pressure of an aneurysm which probably arises from the transverse, or descending, portion of the arch and extends backwards. Other signs of sympathetic irritation or paralysis, such as unilateral flushing and sweating, or pallor and dryness, of the face and neck on the affected side may be detected, but are less conspicuous and less frequent.

Paralysis of one-half of the diaphragm may result from compression of the phrenic nerve. In exceptional cases there are other manifestations of late syphilitic disease of the spinal cord. Of these *tabes dorsalis* is the most frequent. In cases of aneurysm, it is rare to find the classic manifestations of *tabes dorsalis* well developed ; but a careful search may elicit some of the signs. Thus, in a man aged thirty-four years, a large aneurysm was bulging between the third and fifth ribs, from the right border of the sternum as far out as the nipple ; the pulse was smaller in the right than in the left radial artery ; the aortic valve was incompetent ; the pupils and knee jerks were normal ; but both Achilles jerks were abolished. Two cases of our series presented a diffuse sclerosis of the spinal cord. One was a man aged fifty-three, who ultimately died by rupture of a saccular aneurysm into the left bronchus. The second was forty-one years of age when he came under our notice, and he was seen at intervals for ten years. The left vocal cord became paralysed after he had been subject to paroxysmal spasm of the laryngeal adductors. Four years later he developed lightning pains, ataxia and spasticity of the lower limbs, and eventually both knee and Achilles jerks became abolished.

IV. Alimentary System.—Dysphagia, the chief symptom, is not infrequent. It is most common in cases of aneurysm of the transverse portion of the arch and of the descending thoracic aorta. The degree of dysphagia is usually variable from day to day, and is often lessened when the patient leans forward and thus reduces the pressure upon the œsophagus. Dysphagia is usually a late symptom ; but it may be the sole complaint, as in one of our patients who died suddenly from hæmorrhage five weeks later. There is seldom complete obstruction to the passage of food through the œsophagus, or the regurgitation of undigested food and of mucus so suggestive of carcinoma of the œsophagus. In the latter disease the pain is more strictly related to deglutition and is generally referred to the region of the sixth costal cartilage. In other cases of aneurysm the main difficulty in swallowing arises through liquid or solid food entering the larynx during deglutition and thus causing choking.

V. Other Systems.—Of the bones, the sternum is most frequently eroded by aneurysm of the ascending or transverse portion of the arch; the dorsal vertebræ by aneurysms of the descending portion of the arch or of the descending thoracic aorta (Fig. 304). The most constant and agonizing pain is that of erosion, and especially rapid erosion, of bone.



FIG. 320.—RADIOGRAM. ANEURYSM OF DESCENDING THORACIC AORTA. ($\times \frac{2}{3}$) (J. R. R.).

II. ANEURYSM OF THE DESCENDING THORACIC AORTA

Aneurysms of the descending thoracic aorta are comparatively infrequent, constituting about one-tenth of all aortic aneurysms, but may attain a large size. They give rise to various symptoms, but to few or no physical signs until they are large. Usually, the earliest symptom is constant, severe

and gnawing pain in the interscapular region, under the left scapula, or elsewhere in the back. The pain may radiate around the chest and into the left hypochondrium owing to implication of the nerve roots. In other cases the pain simulates that of lumbago. If the aneurysm is developing markedly to the right, the pain may be referred to the right side of the chest, especially posteriorly, or may radiate around the chest into the right hypochondrium. In one of our cases the pain in the latter region simulated that of biliary colic.

The patient has usually been under treatment for months, even for years, before the increasing pain arouses the suspicion of an aneurysm. In the later stages, when the aneurysm is eroding the dorsal vertebræ, the pain is very severe, and ultimately paraplegia may ensue. The aneurysm may cause dyspnœa by compression of the lung, and dysphagia by compression of the œsophagus.

Unless the aneurysm is of large size, there are seldom any abnormal physical signs. The earliest sign is often impairment of resonance in the left side of the chest posteriorly, particularly in the interscapular region; over this area the cardiac sounds may be heard more clearly than in health. The radial pulses and the pupils are equal, and the vocal cords are unaffected. Later, the aneurysm may cause retraction, or bulging, of the left side of the chest, with considerable dullness and with impairment of the breath sounds. Moreover, the aneurysm may be felt pulsating in some part or other of the chest, and may displace the heart to the right or left. The recognition of an aneurysm of the descending thoracic aorta is often attended by much difficulty, as in the following case. A man aged fifty-three, and weighing eighteen stones, took ill suddenly. The left side of the chest, which was slightly enlarged and yielded a dull note, absence of breath sounds and of vocal resonance, was aspirated three times at the angle of the left scapula. On each occasion about twenty ounces of pure blood were withdrawn. The autopsy, performed by one of us a week later, showed that an enormous fusiform aneurysm of the descending thoracic aorta compressing the left bronchus had been aspirated. The whole of the left lung was in a state of grey hepatization.

It is in the diagnosis of these aneurysms that radiographic

examination is of the utmost assistance, and that oblique illumination (*see* p. 559) should never be omitted. If an aneurysm of the descending thoracic aorta ruptures, it is usually into the left pleura or œsophagus, less often into the left lung. In one of our cases, a man aged forty-two, a large aneurysm, which had caused agonizing pain at intervals for two years and which had eroded the fourth to the eighth dorsal vertebræ and the heads of the corresponding ribs on the left side, ruptured into the right pleura. In another, who died at the age of fifty-four, there were two aneurysms. One, a saccular aneurysm, eroding the bodies of the seventh, eighth and ninth dorsal vertebræ, ruptured into the left bronchus; the second was a dissecting aneurysm eroding the bodies of the eleventh and twelfth dorsal vertebræ.

III. ANEURYSM OF THE ABDOMINAL AORTA

Aneurysms of the abdominal aorta are decidedly rare, for they do not constitute more than 1 in 20 or 1 in 30 of all aortic aneurysms. The aneurysm is usually saccular, originates near the cœliac axis, and attains a large size. Upon the anterior wall of the abdomen, and most frequently in the epigastrium, it may eventually form a large, circumscribed protrusion (Fig. 321), which is felt to be expansile with each beat of the heart.

Before it attains this size, however, it has caused symptoms which may be protean in character. Constant pain, with severe paroxysmal exacerbations, is the predominant symptom. The pain may be referred to any part of the abdomen, to the back or to the loins. It may simulate disease of the hip or sacro-iliac joint; compression of the lumbar and sacral nerve-roots may cause the pain to radiate into the lower limbs; by compression of the small or of the large intestine the aneurysm may cause symptoms suggesting chronic intestinal stasis or acute obstruction.

Less often the patient refers all his symptoms to the urinary system, as in the following case, for which we are indebted to Mr. J. W. Dowden.

The patient, aged fifty-seven, had complained for six years of increased frequency of micturition, and for three years of difficulty in

starting micturition and of pain. When seen by Mr. Dowden, the attacks of pain and of strangury were usually nocturnal and were relieved only by morphia. The pain radiated from the lower lumbar and sacral regions to the lower part of the abdomen and to the perineum. The quantity of urine passed on each occasion was small ; it

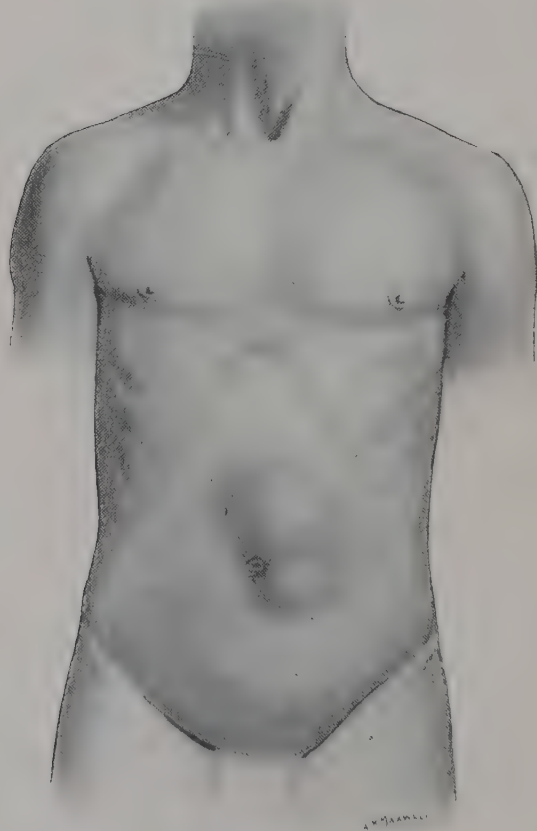


FIG. 321.—SACULAR ANEURYSM OF THE ABDOMINAL AORTA PROJECTING ON THE WALL OF THE ABDOMEN.

contained a little albumin but no blood. The prostate and bladder were ascertained to be healthy and radiographic examination revealed nothing abnormal. A fortnight later the patient became suddenly collapsed and died owing to retroperitoneal rupture of an aneurysm which was situated about two inches below and bulging towards the

left kidney. The aneurysm was adherent to and compressed the upper end of the left ureter, the mucosa of which was infiltrated with blood.

Paraplegia, by erosion of the vertebræ and compression of the spinal cord, is a rare complication.

On palpation of the abdomen, the definitely expansile character of a circumscribed tumour, characteristic of abdominal aneurysm, is not always elicited. The palpable, diffuse throbbing of the abdominal aorta in its whole course, so common in thin neurotic and neurasthenic, and often anæmic, women, is not infrequently mistaken for an aneurysm. These patients complain mainly of the throbbing, seldom if ever of pain, whereas pain is the urgent symptom in aneurysm of the abdominal aorta.

Compression or thrombosis of the distal part of the aorta, or of one or both iliac arteries, may develop with resultant loss of the pulse in one or both femoral arteries; and embolism may occur in the femoral artery with resultant gangrene of the lower limb, or in the superior mesenteric artery. Œdema of the abdomen and of both lower limbs points to compression of the inferior vena cava.

COURSE OF AORTIC ANEURYSM AND MODE OF DEATH

Aortic aneurysms usually enlarge slowly, and the patient suffers for several, or for many, years before death ensues. In illustration of this, we recall the case of a lamp-lighter who came under treatment at the age of thirty-nine for an aneurysm of the ascending aorta. His occupation as keeper of one of the Edinburgh monuments involved his ascending frequently in the course of the day to a considerable height; nevertheless he survived for twelve years.¹

The fatal termination is usually due, not to rupture of the aneurysm, but to cardiac failure. This may develop gradually, with progressive asthenia, or may be sudden. In other cases death is due to terminal pneumonia or to gangrene of the lung, the latter event being most liable to supervene when there is compression of the root of the lung. In other patients death is due to concomitant disease of a chronic nature,

¹ Gibson, G. A., *Diseases of the Heart and Aorta*, Edin. and Lond., 1892, 880.

affecting usually the arteries or the kidneys, or to acute inter-current disease which may have no etiological relation to the aneurysm.

Death by rupture is not common. For a thoracic aneurysm to terminate by fatal rupture on the surface of the body is very rare. In over forty years' service as a hospital physician, Gairdner¹ did not see a single case. If rupture does occur it is usually into the trachea, bronchus or œsophagus. The rupture may be heralded by the aneurysm "weeping" for days or months because of erosion of the mucosa of the trachea, bronchus or œsophagus. When rupture occurs, whether it be internal, for example into the pericardium or pleura, or into the mediastinal or retroperitoneal tissues, or whether it be external by way of the trachea, œsophagus or stomach, there is sudden collapse and speedy death. The patient seldom survives a copious hæmorrhage. Nevertheless we have seen a man survive the loss of half a wash-hand basinful of blood, owing to an aneurysm bursting into a bronchus, until a still more copious and fatal hæmorrhage ensued a few days later. The celebrated surgeon Liston lived for five months after the first gush of arterial blood, and Gairdner² recorded a case in which hæmoptysis recurred for six or seven years before it proved fatal. One of the most remarkable examples of non-fatal rupture is that recorded by Syme³ of an aortic aneurysm which burst upon the upper part of the chest wall. The man held a bowl at arm's length to receive the contents, as he thought, of a "bloody boil," squeezing it meanwhile with his chin. When he had lost about a quart of blood in a stream somewhat thicker than a quill, he fainted, the bleeding stopped and never recurred. Four months later he died of typhus.

DIAGNOSIS

Although the recognition of an aneurysm of the aorta may be an easy matter, it may at other times be attended with great difficulty, especially if the aneurysm does not occasion

¹ Gairdner, W. T., *Allbutt's System of Medicine*, Lond., 1899, vi., 384.

² Gairdner, W. T., *Clinical Medicine*, Edin., 1862, 516.

³ Syme, J., *Monthly Journ. Med. Sci.*, 1850, x., 89.

any pulsation of the thoracic wall and if a radiographic examination is not available.

All the relevant facts should be considered, and due weight laid on the patient's sex, age, and occupation, on the history of spirochætal infection and on the Wassermann reaction. The diagnosis should never be based upon one symptom or sign, however suggestive it may be. Thoracic pain, for example, even though constant and severe, may be due to many other causes; an abnormal area of dullness may be due to disease of the lung, to pleural or pericardial effusion or to mediastinal tumour; inequality of the radial pulses may result from abnormal distribution of one radial artery; irregularity in the size of the pupils may be due to tabes dorsalis or to local causes such as iritis; and paralysis of the left vocal cord may arise from compression of the left recurrent laryngeal nerve by enlarged mediastinal glands, intrathoracic tumour, pleurisy, or in rare cases in mitral stenosis (*see* p. 284). It is also important to determine the sequence of development of the symptoms, because the pain of aortic aneurysm almost invariably precedes dyspnœa, dysphagia, or cough, and hæmoptysis is a late symptom.

In every case, and especially when the physical signs are obscure, radiographic examination is of the utmost service. It is often absolutely essential in the diagnosis of aneurysms of the descending thoracic aorta.

1. Tumours and Abscess of the Chest Wall sometimes simulate aortic aneurysm, especially if they are developing from, and are causing a protrusion forwards, of the upper part of the sternum. One of our cases was thought to be a retro-sternal tuberculous abscess of the manubrium; another proved to be an abscess of the anterior end of the second left rib. In these conditions the marked local tenderness, and the absence of pulsation and of pressure symptoms, are usually notable. F. Macrae, however, saw a case in which a very vascular, pulsatile and expansile sarcoma, perforating the upper part of the right side of the chest, simulated aneurysm closely, and the diagnosis was only made at the autopsy. Before an aortic aneurysm protrudes upon the chest wall it has attained a large size and has already caused other striking symptoms.

2. Other Diseases of the Circulatory System.—The pain of aortic aneurysm is more constant than that experienced by sufferers from myocardial or valvular disease, is less definitely associated with physical exertion, and is referred to the manubrium sterni or to the upper part of the chest, and more often to the right side, rather than to the apical region.

The pain of early *syphilitic aortitis* closely resembles that of an aneurysm of the ascending aorta. The patient is of the age and sex in which aortic aneurysm is most frequent; the history of infection usually dates back about ten or twenty years, as it also does in aneurysm; the second sound at the aortic area may be loud and ringing or may be replaced by a diastolic murmur of aortic incompetence; but there is no abnormal area of dullness or pulsation, nor are there other physical signs indicating aneurysm. Radiographic examination may reveal a moderate degree of broadening, and increased opacity, of the aortic shadow, and excludes with certainty the presence of a saccular or fusiform aneurysm.

The abnormal area of percussion dullness caused by an aneurysm is seldom liable to be mistaken for that of cardiac enlargement or of a pericardial effusion.

Aortic Incompetence may simulate an aneurysm of the ascending or transverse portion of the arch. Reference has already been made to the pain so often accompanying this valvular lesion and its associated syphilitic aortitis. Moreover, in aortic incompetence the ascending aorta is always somewhat dilated, and this may cause a pronounced systolic thrill in the upper sternal region, an area of dullness to the right of the manubrium sterni, and forcible pulsation in the episternal notch. Further, we know that incompetence of the aortic valve is not infrequently associated with aortic aneurysm. In these cases the recognition of a pulsatile tumour within the chest, or of definite signs of compression of the trachea, the left recurrent laryngeal nerve, or other structures, is the only safe indication, apart from X-ray examination, of aortic aneurysm. By radioscopy of the patient affected with aortic incompetence we see the shadow of the ascending aorta, enlarged, bulging outwards towards the right, and pulsating freely, but the enlargement is of moderate degree and is diffuse, not globular (Fig. 314).

The differentiation of abdominal aneurysm from a throbbing abdominal aorta has been discussed on page 575.

3. Diseases of the Respiratory System.—*Tumours of the lung and of the mediastinum* often simulate aortic aneurysm very closely, and the differentiation therefrom may be a matter of considerable difficulty, especially if the tumour has a communicated pulsation from the aorta. Whereas aortic aneurysm is most frequent in males between the ages of thirty and forty-five, who have been infected with syphilis, mediastinal tumours occur in both sexes, and at any age, and are independent of antecedent syphilitic infection. In cases with a primary carcinoma of the stomach, breast, or uterus, or with a primary sarcoma of bone, the onset of thoracic pain, cough and hæmoptysis, suggesting metastasis within the lung, is usually confirmed by the subsequent development of an area of dullness in one lung, and often of a pleuritic effusion, which yields a sanguinolent fluid. In lymphadenoma (Hodgkin's disease) and in lymphosarcoma, however, the earliest of all symptoms may be those of mediastinal tumour causing pressure symptoms. Nevertheless dyspnœa, cough and frequent hæmoptysis are comparatively early symptoms, pain is comparatively late. If one side of the chest is especially involved, it is more often enlarged, whereas in aortic aneurysm it is more often retracted. The area of dullness may conform closely to that which is frequently observed in aneurysm; the part is, however, seldom pulsatile. Enlargement of the spleen and liver, and of cervical, axillary and inguinal glands should be looked for. Examination of the blood, even apart from the Wassermann reaction, may be of aid in diagnosis, because a pronounced degree of anæmia, a leucocytosis, or a glycogen reaction are more frequent in association with tumour than with aneurysm.

Radiographically the diagnosis is comparatively easy, for a tumour mass has not the circumscribed, sharply defined outline of an aneurysm, but invades one lung, or both, diffusely, and its shadow can be seen distinct from that of the aorta.

Pulmonary tuberculosis may be simulated by an aortic aneurysm compressing a main bronchus, or one of its large branches, thereby causing bronchiectasis and collapse of one

lung or one lobe. Pain is, however, a more constant, and hæmoptysis a less frequent symptom ; the sputum is usually foetid, and does not contain tubercle bacilli. Gangrene of the lung may supervene.

Very rarely an aneurysm is simulated by an encysted empyema to which an impulse is communicated from the heart or aorta.

Asthma may be simulated, particularly by a small aneurysm of the transverse portion of the arch compressing the trachea. The onset of the paroxysms in middle life, the absence of their seasonal periodicity so common in pollen asthma, and of any anaphylactic idiosyncrasy to certain proteins of the food, the absence of eosinophilia, and the failure of adrenalin and of belladonna to arrest the paroxysms, are all of importance in diagnosis. A careful search for signs of aneurysm should not be omitted.

Paroxysmal hysterical dyspnœa, with laryngeal stridor, in patients of middle age who are affected with valvular disease, may suggest aortic aneurysm. In one woman, aged forty-nine, the simulation was very close, but radiographic examination proved the aorta to be healthy.

The dyspnœa resulting from tubercle of the larynx can be excluded by laryngoscopic examination.

4. Diseases of the Alimentary System.—Carcinoma of the œsophagus, causing dysphagia, may lead to paralysis of the left recurrent laryngeal nerve, and occasionally of the right, and thus simulate aneurysm of the aorta. But in the latter disease dysphagia is rarely an early symptom ; the pain is more constant and less definitely related to the swallowing of food ; emaciation is less rapid, and signs of compression of other structures probably co-exist. Radiographically the appearances of the two diseases are distinct. Examination by means of the œsophageal bougie or the œsophagoscope must never be attempted until the possibility of aortic aneurysm has been excluded, because of the risk of rupturing the aneurysm.

Visceroptosis very rarely leads to error in diagnosis, but when the diaphragm is situated at an unusually low level and does not adequately support the heart, the latter may

with each beat tug down the aorta and thus cause tracheal tugging.¹ We have never observed this.

5. Of Diseases of the Nervous System those most liable to be mistaken for aortic aneurysm are those in which paralysis of a vocal cord or of the cervical sympathetic arise, as discussed on page 569. Complete recurrent laryngeal paralysis of one cord may be simulated when there is fixation of the cord by perichondritis of the arytenoid cartilage; this gave rise to difficulty in one of our cases, a man aged twenty-eight; but the cord, although fixed, had not the true cadaveric form. Hysterical dysphagia and aphonia seldom present any difficulty in diagnosis. The complete dysphagia occurring after occlusion of one posterior-inferior cerebellar artery is associated with other signs of a unilateral bulbar syndrome, namely asynergia of the homo-lateral limbs and dissociated anæsthesia of the contra-lateral side of the body. Chronic meningitis, a tumour of the spinal cord, and tuberculous or malignant disease of the vertebræ may be simulated if the aneurysm is compressing intercostal nerves or nerve-roots, and causing marked girdle pain. Paraplegia, due to erosion of dorsal vertebræ, may arise as a late complication of an aneurysm of the descending thoracic, or abdominal, aorta.

6. Disease of the Vertebræ—sarcoma, tubercle, rheumatoid arthritis, etc.—may be mistaken for a deep-seated aneurysm. In these affections of the vertebræ, however, pain on movement of the back, tenderness localized to one or more of the vertebræ, and rigidity of the spine are notable symptoms; there is often a well-defined prominence at the affected region; and radiograms are distinctive.

PROGNOSIS

The prognosis in aneurysm of the aorta is invariably unfavourable. The detection by radiographic examination of a small aneurysm within the thorax, causing no symptoms, is, however, by no means an infrequent event, and cases of this nature may survive for ten or even twenty years, and die from some intercurrent disease. *Post-mortem* statistics also show the

¹ Wenckebach, K. F., *Samml. klin. Vortr.*, 1907, No. 465, 466.

frequency of aortic aneurysms which had remained entirely latent.

In general, the prognosis rests upon the size of the aneurysm, the rapidity of its growth, and the degree of compression it exerts upon important structures; and further upon the nutrition of the heart muscle, the presence or absence of disease of the aortic valve, and upon the patient's financial circumstances. A large aneurysm causing symptoms of compression is always of great gravity, but a small aneurysm compressing the trachea or left bronchus is no less grave. In comparatively few cases does death ensue by rupture (*see* p. 576), but when an aneurysm begins to "weep," the end is not far distant. The outlook is more favourable in patients who can afford to lead a life of enforced rest than in those who, because of straitened financial circumstances, cannot desist from work.

Temporary alleviation of all, or of many, distressing symptoms is often obtainable by means of treatment, but in the more serious cases palliation alone is possible.

TREATMENT

1. *Prophylactic Treatment.*—Every case of syphilitic aortitis, with or without disease of the aortic valve, should have a thorough course of anti-syphilitic treatment, first with one of the organic arsenical preparations, and thereafter with mercury and potassium iodide for at least one year. If the aortitis is still in an early phase, material benefit may be anticipated from that course of treatment, inasmuch as it lessens the risk of aneurysm developing. At the same time the patient should, as far as possible, cease from undertaking arduous labour, and should seek an occupation as light as possible.

2. *Curative Treatment.*—Once an aneurysm has developed the aim of treatment is to promote thrombosis within the sac and thus to arrest the growth of the aneurysm. With this aim in view, it is desirable to reduce the rate of contraction of the heart, and to lower the arterial blood-pressure, and thereby to lessen the velocity of the blood and the pressure within the aneurysm. Recumbence alone may lessen the rate of the heart by twenty-one thousand beats per day.

Complete rest, mental as well as physical, is therefore of paramount importance. No other line of treatment is of equal avail. A few days of complete recumbence in bed may bring temporary relief from most of the patient's sufferings, but the enforced rest and skilled nursing must extend over a period of two or three months if any lasting benefit is to accrue. It is often necessary to revert from time to time to this mode of treatment when symptoms recur.

Classic writers, following Valsalva, emphasized the importance not only of rest but of restricting the patient's diet, even to the point of semi-starvation, of limiting his intake of fluid to the utmost, and of venesection. The dietetic treatment advocated by Tufnell,¹ though less rigorous, permitted only ten ounces of solid, and eight ounces of liquid food, in the twenty-four hours. It has still to be proved, however, that the total volume of the blood can materially be lessened by this stringent method of treatment; and equally favourable results are obtainable by a moderate limitation of the solid and fluid intake.

In addition to rest and dieting, potassium iodide is usually of much benefit. Although the administration of this drug is not invariably followed by the brilliant results recorded by Balfour,² to whose advocacy its wide adoption in aortic aneurysm is due, nevertheless it is still the most potent of all medicinal substances in the treatment of aortic aneurysm. Its administration in doses of ten or fifteen grains thrice daily is often attended with striking amelioration of pain, and with lessening of other pressure symptoms. The beneficial action of the drug is probably to be explained not by its lowering the blood-pressure, but by its well-known power of promoting the absorption and healing of syphilitic granulation tissue. It is usually advisable to administer mercury at the same time. In selected cases presenting neither grave cardiac enfeeblement nor advanced renal disease, a series of intravenous injections of one or other of the organic preparations of arsenic should be given in the hope of arresting the spread of the

¹ Tufnell, J., *The Successful Treatment of Internal Aneurism*, Lond., 1875, 2nd Edit.

² Balfour, G. W., *Clinical Lectures on Diseases of the Heart and Aorta*, Lond., 1898, 3rd Edit., 437.

syphilitic aortitis ; it matters little which preparation is employed. It is well, however, to begin with a small dose (0.3 gramme) and to increase it gradually once a week until the full dose is given. Subsequently a prolonged course of mercury and iodide is always indicated.

The employment of calcium salts in the expectation of increasing the coagulability of the blood, and of thereby promoting thrombosis within the aneurysmal sac, is seldom attended with any definite benefit.

The results of surgical treatment such as the introduction of metallic wire, and electrolysis, both leading to the formation of red thrombi within the sac, have almost invariably been disappointing. In selected cases, however, Sir William Macewen's method of needling the inner wall of the aneurysmal sac is more hopeful. In a case which came under observation while one of us was house-physician in Dr. Tennent's wards this method was attended by a good result. The aneurysm was situated to the right of the sternum and its projection above the ribs was as large as the half of an orange. Needles were introduced on several occasions for varying periods, on one occasion for nearly twenty-four hours. One needle broke *in situ*. The sac, which had previously been increasing in size and causing thinning of the skin, underwent consolidation and shrinking. Five years later the patient still maintained good health.

Few abdominal aneurysms are of such a nature as to permit of surgical treatment ; in a few instances they have been dealt with successfully by means of compression applied proximally to the sac.

3. *Symptomatic Treatment.*—For the relief of symptoms complete rest and the administration of potassium iodide are often the most useful means at our disposal. Pain, however, and particularly that due to erosion of bone or to compression of nerves, may necessitate the administration of opium, morphia, or heroin. In promoting sleep, one-twelfth or one-sixth of a grain of morphia or heroin is more efficacious than other hypnotic or analgesic remedies. Morphia, if needed, should never be withheld ; in some patients large doses are needed daily. Local applications are practically valueless for the relief of pain ; and the application of local pressure

to an aneurysmal sac which is protruding upon the chest wall, in the hope of preventing its rupture externally, is never justified. Venesection may afford temporary relief in cases presenting urgent signs of tracheal compression. Even although the lowering of the total volume of the blood thus effected is of short duration, it may tide the patient over a dangerous crisis.

INDEX

- a* wave, 61
- Abdominal aorta, aneurysm of, 573
- Abductor paralysis of larynx, 568
- Aberrant ventricular complex, 104
- a-c* interval, 64
 - decrease of, 105, 211
 - events during, 64, 115
 - increase of, 105, 115
 - normal, 64
 - prolonged, 105, 115
- Abscess of bundle, 122
 - chest simulating aneurysm, 577
 - myocardium, 47
- Acceleration of heart, by adrenalin, 78
 - by atropin, 78
 - by exercise, 24, 78
 - in fever, 79
- Acidosis, 28
- Action currents, 66
- Adams-Stokes syndrome, 127
 - atropin, action of, in, 133
 - causation of, 130
 - cerebral anæmia in, 130
 - diagnosis of, 132
 - heart-block and, 127, 140
 - in acute lesions of bundle, 130
 - prognosis in, 132
 - pulse in, 130
 - standstill of ventricles in, 130
 - sino-auricular block and, 140
 - ventricles in, 130
- Adherent pericardium, 428
- Adrenalin, action of, on arteries, 491
 - blood-pressure, 481
 - sympathetic, 24
 - T* deflexion, 72
 - vagus, 23
- arterio-sclerosis and, 481
- extrasystoles and, 88
- tachycardia and, 78, 79
- Age and prognosis, 355
- Age incidence of aneurysm of aorta, 547
 - angina pectoris, 391
 - extrasystoles, 89
- Age incidence of fibrillation, 175
 - flutter, 157
 - valvular disease, aortic, 269
 - mitral, 269
- Air, volume of, breathed, 29
- Albuminuria, 290, 506
- Albuminuric retinitis, 517
- Alcohol in arterio-sclerosis, 529
 - in cardiac failure, 386
- Alcoholism, arterio-sclerosis and, 525
 - dilatation of heart in, 153
- Alkali reserve, 27
 - lowering of, 28
- Alkalosis, 29
- Alternation of pulse, 143
 - auricular flutter and, 164
 - arterial tracing of, 148
 - diagnosis of, 148
 - digitalis and, 150
 - electrocardiograms of, 149
 - etiology of, 146
 - extrasystoles and, 111, 147
 - frequency of, 146
 - latent, 148
 - nature of, 145
 - prognosis in, 150
 - tachycardia and, 150, 164
- Amines and blood-pressure, 528
- Ammonium salts in arterio-sclerosis, 536
- Amyl nitrite in angina pectoris, 397
- Anacrotic pulse, 335
- Anæmia, dilatation of heart in, 154, 291
 - in endocarditis, 387
 - murmurs in, 154, 316
 - myocardium in, 37, 154
- Anaphylactic heart-block, 115
- Anastomosis of coronary arteries, 3, 40
- Aneurysm of aorta, 544
 - abdominal aorta, 573
 - age incidence of, 547
 - aortitis and, 546
 - arch of aorta, 551
 - bronchus, compression of, by, 566
 - cough in, 566

Aneurysm of arch of aorta, dullness of, 556
 dysphagia in, 570
 dyspnoea in, 564
 hæmoptysis, 566
 impulse of, 555
 laryngeal paralysis in, 567
 ocular signs of, 569
 Oliver's sign in, 567
 pain of, 552, 572, 573
 pulsation of, 554
 pulse in, 558
 inequality of, 558
 pupils in, 569
 radiographic signs of, 558
 rupture of, 575
 signs of, 551
 sounds over, 557
 stridor in, 564, 580
 sympathetic cord, com-
 pression of, by, 569
 symptoms of, 551
 trachea, compression of, by,
 564, 567
 venous compression by, 554
 blood-pressure and, 542
 bone, erosion of, by, 571
 Broadbent's classification of, 552
 course of, 575
 death in, 575
 diagnosis from asthma, 580
 empyema, 580
 larynx, disease of, 580
 lung, tumours of, 579
 lymphadenoma, 579
 mediastinal tumours, 579
 nervous system, diseases of,
 581
 œsophagus, carcinoma of, 580
 tuberculosis, 579
 vertebræ, disease of, 581
 visceroptosis, 580
 dissecting, 549
 etiology of, 544
 fusiform, 549
 infective, 245, 544
 mesaortitis in, 486, 544, 546
 occupation, 548
 prognosis of, 581
 pupils in, 569
 rupture of, 575
 saccular, 549
 sex incidence of, 547
 site of, 549
 size of, 549
 syphilis and, 544
 thoracic aorta, descending, 571
 treatment of, 582
 iodide in, 583
 rest in, 583

Aneurysm of aorta, surgical treat-
 ment of, 584
 venesection in, 585
 Wassermann reaction in, 546
 Aneurysm of ductus arteriosus, 466
 heart, 54, 365
 retinal arteries, 515
 Angina pectoris, 388
 blood-pressure in, 391
 diagnosis of, 395
 defective contractility and,
 146
 etiology of, 390
 incidence of, 391
 pain of, 388
 pathology of, 393
 prognosis of, 396
 referred pain of, 389, 395
 toxic causes of, 392
 treatment of, 397
 varieties of, 388
 Anisocoria, *see* Pupils
 Anoxæmia, 29
 Ante-mortem clots, 193, 293
 Aorta, aneurysm of, *see* Aneurysm
 development of, 454
 rupture of, 394, 575
 syphilis of, 485, 544
 Aortic aneurysm, *see* Aneurysm
 incompetence, 295, 336
 blood-pressure in, 338
 compensation of, 297, 339
 Flint's murmur in, 338
 murmurs in, 295, 337
 pulse in, 338
 signs of, 336
 syphilis and, 260, 270
 stenosis, 333
 compensation of, 297, 335
 murmurs in, 317, 334
 signs of, 333
 valve, acute endocarditis of, 215
 mechanism of, 295, 316
 rupture of, 261
 valvular disease, 252
 arterial disease and, 267, 271
 cardiac rhythm in, 352
 cardio-vascular degenera-
 tion and, 267
 Cheyne-Stokes breathing in,
 281
 chorea and, 273
 coronary arteries in, 257, 278
 dyspnoea in, 280, 301
 endocarditis, acute, and,
 258, 270
 etiology of, 257, 268
 incidence of, 269
 kidneys, disease of, in, 267
 mitral incompetence in, 384

- Aortic -valvular disease, morbid**
 anatomy of, 252
 myocardial failure in, 353
 cedema in, 287
 pain in, 286
 palpitation in, 284
 prognosis in, 350
 pulse in, 335, 338
 renal disease and, 262
 signs of, 332
 strain, mechanical, and, 261, 271
 symptoms of, 295, 300
 causes of, 275, 299
 syphilis and, 260, 270
 treatment of, 370
 ventricle, left, in, 70, 297
- Aortitis, infective, 491, 544**
 syphilitic, 260, 485, 546
- Apex impulse, fixation of, 432**
 inverted, 309, 432
 normal, 307
 tracings of, 58, 310
- Apnœa, *see* Cheyne-Stokes breathing**
- Arborization block, 138**
- Arches, primitive aortic, 455**
- Arrhythmia, respiratory, of heart, 84**
 sinus, 83
- Arsenic in aneurysm of aorta, 583**
 syphilis of heart, 386
- Arterial diseases, 469**
 arterio-sclerosis, 472
 atheroma, 482
 calcareous degeneration, 489
 diffuse lesions, 472
 endarteritis obliterans, 487
 etiology of, 476, 490
 extrasystoles in, 90
 focal lesions of, 482
 mesarteritis, 485
 ocular manifestations in, 514
 prognosis of, 519
 symptoms of, 495
 syphilitic, 485
 sounds, *see* Sounds
 tracings, 59
- Arteries, coronary, *see* Coronary arteries**
 diseases of, 469
 focal lesions of, 482, 490
 retinal, 514
 structure of, 470, 471
 vascular arrangements of, 471
- Arterio-sclerosis, 472**
 adrenalin and, 481, 491
 albuminuria in, 506
 alcoholism and, 525
 and cardiac hypertrophy, 499
 apex impulse in, 499
 blood-pressure in, 142, 535
- Arterio-sclerosis, bronchitis in, 510**
 cardiac failure in, 511, 533
 cerebral symptoms in, 512
 constipation in, 530
 diagnosis of, 499
 diet and, 528, 530
 diuretics and, 530
 drugs in, 532
 elastic fibres in, 473
 emphysema in, 510
 enteric fever and, 481
 etiology of, 476
 exercise and, 530
 hæmorrhage in, 525
 iodide in, 543
 lead-poisoning and, 481
 massage in, 531
 miliary aneurysms and, 515
 morbid anatomy, 472
 ocular manifestations in, 514
 oral sepsis in, 525
 polycythæmia and, 479
 pulse in, 499
 purgatives in, 530
 renal changes in, 506
 retina in, 514
 sepsis and, 525
 shortness of breath in, 510
 sounds of heart in, 506
 symptoms of, 496
 treatment of, 521
 uræmia in, 510, 539
 valvular disease in, 507
 vaso-dilator drugs in, 535
 visceral lesions in, 497
 viscosity of the blood in, 478
- Arthritis, endocarditis and, 223**
 pericarditis and, 412
- Ascites in mediastino-pericarditis, 430**
 valvular disease, chronic, 287
- Asphyxia, auriculo-ventricular block and, 115**
 nodal rhythm and, 199
- Asthma, cardiac, *see* Dyspnœa**
- Atheroma, 482**
- Atresia of pulmonary artery, 161**
- Atria, *see* Auricles**
- Atrio-ventricular, *see* Auriculo-ventricular**
- Atrophy of heart muscle, 32**
 ischæmic, 32
- Atropine, action of, 22, 78**
 in Adams-Stokes syndrome, 133
 in heart-block, 115, 133
 in nodal rhythm, 201, 205
 in sinus irregularity, 86
 on vagus, 22
- Auricles, contraction of, 61, 68**

- Auricles, fibrillation of, *see* Fibrillation, 174
 flutter of, *see* Flutter, 156
 musculature of, 1
 thrombi in, 193, 293
- Auricular canal, 5, 453
 contraction, 61, 68
 deflexion, 68, 104, 166, 211
 fibrillation, *see* Fibrillation, auricular, 174
 flutter, *see* Flutter, 156
- Auriculo-ventricular block, *see* Block bundle, *see* Bundle
 conductivity, *see* Conductivity dissociation, 121
 extrasystoles, 105
 node, lesions of, 122, 200
 site of, 6
 valves, opening of, 61
- Axis of heart, electrical, 70
- b* wave, *see* *h* wave
- Baillie's pill, 385
- Baths, Turkish, in arterio-sclerosis, 530
- Bicarbonate of blood, 27
- Bigeminal pulse, 97
- Bigeminy, true, 109
- Block, arborization, 138
 auriculo-ventricular, 118
 acute, 121
 Adams-Stokes syndrome in, 127
 adrenalin and, 114, 115
 anaphylactic, 115
 asphyxial, 115
 atropin in, 115
 auricular contractions in, 114, 118
 chronic, 122, 124
 clinical signs of, 124
 complete, 121
 auricular fibrillation and, 190
 flutter and, 168
 ventricular rate in, 121
 congenital, 460
 diagnosis of, 131
 digitalis in, 115, 117, 133
 etiology of, 113, 122
 experimentally, 113
 functional, 114, 121
 grades of, 115
 in acute infective disease, 124
 congenital heart disease, 460
 diphtheria, 460
 endocarditis, 124
 jugular pulsations in, 124
- Block, auriculo-ventricular, morbid anatomy of, 122
 partial, 118
 digitalis and, 120
 in auricular fibrillation, 182
 in flutter, 168
 pulse in, 120
 prognosis of, 132
 prognosis in, 132
 pulse in, 118, 131
 Stokes-Adams syndrome in, 127
 symptoms of, 124
 syphilis and, 123
 treatment of, 133
 vagal stimulation and, 115
 ventricular rate in complete block, 121
 branch, functional, 136
 organic, 133
 electrocardiograms of, 134
 intraventricular, 138
 sino-auricular, 140
- Blood, arterial, 29
 CO₂ of, 27, 29
 cultures in acute endocarditis, 226, 228
 in dyspnoea, 27
 oxygen saturation of, 29
 plasma, 27
 -pressure, diastolic, 338
 digitalis and, 378
 in aneurysm, 542
 in angina pectoris, 390, 391
 in aortic incompetence, 338
 in aortic stenosis, 335
 in arterio-sclerosis, 142, 535
 in mitral disease, 330, 542
 in nephritis, 476, 533
 in oedema, 330
 in tachycardia, 208
 intracranial, and bradycardia, 82
 systolic, 338
 residual, 25, 153
 -sugar, in tachycardia, 79
 -urea, 291
 venous, 29
 viscosity of, 478
 Wassermann reaction of, in aneurysm of aorta, 546
 aortic valvular disease, 271
- Bradycardia, nodal, 205
 sinus, 81
- Branches of bundle, 8
 block of, 133
 severance of, 123, 133
- Breathing, Cheyne-Stokes, 85, 149, 281, 511

- Breathlessness, *see* Dyspnoea
 Bright's disease, *see* Renal disease
 Broadbent's classification of aortic aneurysms, 552
 sign, 433
 Bronchitis in aortic disease, 301
 arterio-sclerosis, 510
 mitral disease, 279, 294, 300
 Bronchus, compression of, by aneurysm, 566
Bruit de diable, 317
 galop, 319
 Roger, 460
 Buffer salts, 27
 Bulbus cordis, 5, 453
 arrested development of, 456, 460
 Bundle, auriculo-ventricular, 8
 compression of, 114
 function of, 19, 113
 in heart-block, 113
 lesions of, 122
 position of, 8
 relations of, 10
 severance of, 113, 123
 site of, 8
 structure of, 15
 branches of, *see* Branches
c wave, 60
 Caffeine in arterio-sclerosis, 536
 in chronic valvular disease, 378, 386
 Calcification, medial, 489
 of arteries, 489
 of bundle, 123
 Calcium salts in aneurysm, 584
 Camphor, 386
 Canal, auricular, 5, 453
 Canter rhythm, 153, 319
 Capillary circulation, 30
 pulse, 338
 Carbon dioxide, *see* CO₂
 Cardiac failure, causes of, 275, 358
 in arterio-sclerosis, 511, 533
 over-strain, 153, 279
 treatment of, 370
 Cardiolysis, 451
 Cardio-pulmonary murmur, 318
 Carditis, rheumatic, *see* endocarditis, 222
 Cerebral symptoms of chronic valvular disease, 289, 305
 Cervical curve in extrasystoles, 96, 104, 106
 fibrillation, 186
 flutter, 164
 heart-block, complete, 125
 partial, 119, 121
 nodal rhythm, 211
 Cervical curve, normal, 59
 sinus tachycardia, 80
 C_H of blood, 27
 Cheyne-Stokes breathing, 85, 149, 281, 511
 Chordæ tendineæ, in acute endocarditis, 221
 in mitral disease, 253
 Chorea and acute endocarditis, 223, 228
 and chronic valvular disease, 273
 and pericarditis, 413
 Circus movement in fibrillation, 177
 in flutter, 159
 Clamping of the bundle, 114
 Claudication, intermittent, 512
 Cloudy swelling of myocardium, 33
 Clubbing of fingers, 245, 320, 462
 CO₂ and dyspnoea, 27, 29
 Coagulation necrosis, hyaline degeneration and, 34
 Colloidal silver in endocarditis, 251
 Compensation in aortic disease, 26, 297, 335, 339
 mitral disease, 26, 328, 331
 prognosis and, 353
 Compensatory pause, 88
 Complex, ventricular, 69
 in branch block, 133
 in extrasystoles, 100, 104
 in heart-block, 126
 normal, 69
 Conduction, *see also* Conductivity
 of stimuli, 113
 Conductivity, 113
 asphyxia and, 115
 atropine and, 115
 auriculo-ventricular, 113
 depression of, 115
 digitalis and, 115
 murmurs in defective, 117
 vagal stimulation and, 115, 117, 133, 141
 Congenital heart disease, 453
 aorta, development of, 454
 arches, primitive aortic, 455
 arrest of development causing, 455
 atresia of pulmonary artery in, 460
 auriculo-ventricular block in, 460
 bruit de Roger in, 460
 chronic valvular disease and, 268
 clubbing of fingers in, 462
 cyanosis in, 461, 467

- Congenital heart disease, diagnosis of, 467
 - endocarditis causing, 455
 - etiology of, 455
 - heart, development of, 453
 - murmurs of, 467
 - prognosis of, 349, 468
 - pulmonary tuberculosis in, 462
 - radioscopy of, 468
 - septal defects in, 455
 - treatment of, 468
- Connective tissue of the heart, 2
- Contraction of heart, 18
- Contractility, 142
 - defective, 142
 - depression of, 142
 - alternating pulse in, 143
 - of parts of heart, 19
 - variations of, 142
- Control of heart, 21
 - mechanical, 24
 - nervous, 21
- Conus arteriosus, development of, 453
 - enlargement of, 466
 - in auricular fibrillation, 184
 - pulsation of, 311, 466
- Convallaria, 386
- Convulsions in Adams-Stokes syndrome, 129
- Coronary arteries, 2
 - anastomosis of, 3, 41
 - angina pectoris and, 393
 - circulation through, 3
 - disease of, 361, 362
 - embolism of, 40, 42
 - in angina pectoris, 393
 - in aortic incompetence, 278
 - ligature of, 41, 175
 - obstruction of, 40, 42, 362
 - sinus, 7
- Corrigan's pulse, 338
- Cough, 283, 566
- Coupled rhythms, 106
 - auricular fibrillation and, 191
 - causes of, 107
 - digitalis and, 107
 - extrasystoles and, 106
 - partial heart-block and, 106
- Currents, action, 66
- Cyanosis in congenital heart disease, 461, 467
 - in pulmonary stenosis, 461
 - in septal defects, 467
- Cyclic breathing, 28, 282
- "D.A.H.," etiology of, 401
- "D.A.H.," hyperthyroidism and, 79
 - post-infective, 404
 - signs of, 400
 - sinus tachycardia and, 78
 - symptoms of, 400, 406
- Death, sudden, 194, 350, 363, 384
 - ventricular fibrillation and, 194
- Deflexions, electrocardiographic, auricular, 68
 - P, 68
 - Q, 69
 - R, 69
 - S, 69
 - T, 72
 - time relation of, 75
 - ventricular, 69
- Degeneration, cardio-vascular, 267
 - of myocardium, fatty, 35, 359
 - granular, 33
 - hyaline, 34
- Derivations, electrocardiographic, 67
- Development of heart, 453
- Dextrocardia, 456
- Diameters of heart in auricular fibrillation, 184
 - normal, 184
- Diastole, duration of, 160
 - prolongation of, 145
 - coupled rhythms due to, 107
 - extrasystoles due to, 92
 - vagal stimulation and, 21, 82
- Diastolic filling of ventricle, 25, 145
 - volume, 151
- Dicrotic notch, 339
 - pulse, 368
 - wave, 339, 340
- Diet in aneurysm of aorta, 583
 - in arterio-sclerosis, 530
 - in cardiac failure, 371
 - Tufnell's, 583
- Digestion and arterio-sclerosis, 528, 530, 532
- Digitaline, 172, 196
- Digitalis, 376
 - alternation and, 150
 - blood-pressure and, 378
 - coupled rhythm due to, 107
 - dosage of, 380
 - heart-block due to, 115, 121, 383
 - in aortic incompetence, 384
 - arterio-sclerosis, 536
 - auricular fibrillation, 195, 379
 - cardiac failure, 376
 - extrasystoles, 383
 - flutter, 171, 382
 - normal rhythm, 383
 - paroxysmal tachycardia, 213, 214

- Digitalis**, in sino-auricular block, 140
 sinus irregularity, 86
 preparations of, 385
 T deflexion altered by, 72
 tonicity and, 155, 377
- Dilatation of heart**, digitalis in, 376
 extrasystoles in, 90
 murmurs in, 318
 physiological, 152
 tonicity, depression of, and, 151
- Diphtheria**, auricular fibrillation in, 176
 " D.A.H. " after, 405
 flutter in, 158
 heart-block due to, 124, 460
 nodal rhythm in, 200
 tonicity depressed by, 154
- Disordered action of heart**, 400
- Dissociation**, auriculo-ventricular, 121
 of myocardium, 55
- Diuretics** in arterio-sclerosis, 530, 536
 in cardiac failure, 378, 386
- Diuretin**, 378, 386
- Dropsy** in aortic valvular disease, 303
 auricular fibrillation, 179
 dilatation of heart, 153, 287
 mediastino-pericarditis, 428
 mitral valvular disease, 303
 myocardial failure, 358
 tonicity, depression of, 153
 valvular disease, chronic, 287, 303
- Dropsy**, treatment of, 375
- Ductus arteriosus**, aneurysmal dilatation of, 466
 closure of, 463
 development of, 455
 patent, cyanosis in, 464
 murmurs of, 464
 pulsus paradoxus in, 465
 radiograms of, 466
 signs of, 464
- Dullness**, normal cardiac, 312
- Duroziez's murmur**, 339
- Dysentery**, " D.A.H. " as sequel of, 405
- Dysphagia** in aneurysm of aorta, 570
 pericarditis, 419
- Dyspnoea**, 27, 280, 300
 acidosis and, 28
 anoxæmia and, 30
 blood in, 27
 cardiac, 28, 280
 Cheyne-Stokes breathing in, 281, 511
 cyclic, 28, 280
 in anæmia, 30
- Dyspnoea** in aneurysm of aorta, 564
 arterio-sclerosis, 510, 533
 aortic disease, 301
 mitral disease, 300
 valvular disease, chronic, 280, 300
 paradoxical pulse in, 149
 paroxysmal, 28, 281, 511
 polycythæmia and, 30
 renal, 28, 282, 511
 toxic, 28, 511
 treatment of, 375
- Ectopic contractions**, 87
 in auricular fibrillation, 184, 191
- Effort syndrome**, *see* " D.A.H. "
- Effusion**, pericardial, characters of, 421
 diagnosis of, 425
 fibrinous, 408
 purulent, 409
 serous, 409
 signs of, 423
 symptoms of, 417
 treatment of, 450
- Electric potential**, changes of, 66
- Electrocardiogram**, normal, 67
 of aortic incompetence, 71, 73
 arterio-sclerosis, 73
 auricular fibrillation, 188
 extrasystoles, 100, 102, 104
 dextrocardia, 456
 flutter, 165, 166
 heart-block, 118, 126
 hypertrophy of left ventricle, 73
 right ventricle, 71
 mitral stenosis, 71
 nodal rhythm, 202, 204, 211
 in angina pectoris, 392
 heart-failure, 72
- Electrocardiograph**, the, 66
- Electrocardiographic deflexions**, 67
 derivations, 67
- Embolism**, cerebral, 304
 coronary, 40, 362
 in acute endocarditis, 244
 mitral disease, 304
 mesenteric, 286
 pulmonary, 283
 renal, 291
- Emotion**, extrasystoles and, 91
 tachycardia and, 78
- Emphysema**, arterio-sclerosis and, 510
- Empyema** simulating aortic aneurysm, 580
- Endarteritis deformans**, 482
 etiology of, 493
 obliterans, 487

- Endocarditis, acute, 215
 aneurysm, infective, in, 220
 aortitis in, 220
 auscultatory evidence of, 233, 241
 bacteriology of, 215
 blood-cultures in, 226
 cardiac muscle in, 222
 cardiac over-action in, 232
 causes of, 223
 chorea and, 223
 cutaneous manifestations of, 245
 diagnosis of, 241
 embolism in, 244
 enteric fever and, 224
 erythema in, 245
 etiology of, 223
 exanthemata and, 225
 extrasystoles in, 232, 244
 fever in, 234, 241
 fingers, clubbing of, in, 245
 gonococcal, 228
 hæmaturia in, 244
 heart-block in, 244
 hemiplegia in, 238
 incidence of, 223
 influenza and, 224
 leucocytosis in, 245
 local infections and, 246
 massage in, 248
 morbid anatomy of, 215
 mural, 220
 murmurs in, 233, 241, 244
 nodal rhythm in, 232
 pericarditis and, 222
 petechiæ in, 245
 pneumonia and, 224
 prognosis of, 246
 pulse in, 230, 243
 rest in, 247
 retinal hæmorrhages in, 237
 rheumatism and, 223, 225
 scarlatina and, 224
 septic foci in, 224, 246
 sinus tachycardia in, 80
 spleen in, 244
 subcutaneous nodules in, 234
 symptoms of, 230
 tonsillitis and, 224
 treatment of, 246
 ulcerative, *see* Malignant
 valves affected in, 228
- Endocarditis, chronic, *see* Valvular disease
 lenta, *see* Endocarditis, subacute
- Endocarditis, malignant,
 bacteriology of, 228
 blood cultures in, 226
- Endocarditis, malignant, causes of, 224
 diagnosis of, 241
 incidence of, 224
 morbid anatomy of, 224
 prognosis of, 246
 rheumatism and, 224
 symptoms of, 235
 subacute bacterial, 229, 240
 nephritis in, 267
- Endocrine glands and tachycardia, 79
- Enophthalmos, 569
- Enteric fever, acute endocarditis and, 245
 arterio-sclerosis and, 482, 492
 "D.A.H." and, 404
- Epigastric pain, 286, 302, 303
 pulsation, 310
- Epilepsy, 132
- Epileptic convulsions, bradycardia and, 85, 129
- Erythræmia, *see* Polycythæmia
- Erythrol tetranitrate, 536
- Excitability of heart, 87
- Exertion, effects of, on,
 blood-pressure, 27
 pulse rate, 78, 353
 work of heart, 24
 volume of heart, 152
- Exophthalmic goitre, alternation in, 150
 auricular fibrillation in, 176
 flutter in, 158
 tachycardia in, 79, 401
- Exophthalmos in aneurysm of aorta, 569
- Extrasystoles, 87
 age incidence of, 89
 alternation after, 145, 147
 arterial tracings of, 93, 94, 97
 auricular, 104
 auricular fibrillation and, 184, 191
 bigeminal pulse from, 97
 blood-pressure during, 94
 causes of, 88
 coupled rhythms from, 106
 definition of, 87
 diagnosis of, 110
 digitalis and, 107, 383
 electrocardiograms of, 100, 102, 103
 emotional origin of, 91
 etiology of, 88
 frequency of, 88
 functional origin of, 90
 heart sounds in, 94
 in arterial disease, 90
 auricular fibrillation, 184, 191

- Extrasystoles, in cardiac failure,
 90
 dilatation of heart, 90
 endocarditis, 90, 232, 244
 fever, 91
 heart-block, complete, 127
 mitral disease, 352
 myocardial disease, 90
 interpolated, 98
 jugular pulsations, 96
 nodal, 105
 palpitation from, 93
 pause after, 88, 94
 posture, effect of, 90
 prognosis of, 110
 pulse in, 93
 bigeminal pulse, 97
 trigeminal pulse, 97
 respiration, effect of, on, 90
 reflex origin of, 91
 series of, 88, 99
 sex incidence of, 89
 significance of, 110
 site of origin of, 88
 supra-ventricular, 96
 sympathetic stimulation causing,
 91
 symptoms of, 92
 toxæmia causing, 91
 treatment of, 112
 trigeminal pulse from, 97
 vagal stimulation causing, 91
 venous pulse, 96
 ventricular 97
 varieties of, 101
- Extrasystolic arrhythmia, *see* Extra-
 systoles
- Failure, cardiac, 358, 506
 prognosis in, 348
 signs of, 358
 treatment of, 370
- Fainting, 290, 306, 358, 400
- Fatigue and branch block, 138
 intraventricular block, 140
- Fatty degeneration of the arteries,
 484
 heart, 35, 359
 causes of, 37, 38
 diffuse, 37
 distribution of, 36
 frequency of, 35
 para-arterial, 37
 patchy, 37
 signs of, 367
 symptoms of, 361
 infiltration of the heart, 34,
 360
 obesity and, 34, 360
- Fermentation, intestinal, 529
- Fever in endocarditis, acute, 234, 241
 pericarditis, acute, 420
- Fibrillation, auricular, 174
 age incidence of, 175
 arterial pulse in, 182
 auricular muscle in, 176
 auriculo-ventricular block
 and, 190, 194
 circus movement and, 177
 clinical features of, 177
 co-existing disease and, 176
 continuous, 177
 death in, 194
 diagnosis of, 192
 digitalis in, 195, 379
 dropsy in, 179
 ectopic contractions in, 184,
 191
 electrocardiograms of, 188
 etiology of, 175
 experimentally, 175
 extrasystoles in, 184, 191
 heart, form of, in, 176, 184
 infarcts in, 193
 mitral stenosis and, 176,
 326
 morbid anatomy of, 176
 murmurs in, 186, 326
 myocarditis and, 176
 paroxysmal, 177
 prognosis of, 192
 pulse deficit in, 184
 pulse in, 182
 quinidine in, 196
 refractory period in, 177
 rheumatism and, 176
 sex incidence of, 175
 symptoms of, 177
 syphilis and, 176
 treatment of, 194
 vagal stimulation and, 177,
 182
 venous pulse in, 186
 ventricular rate in, 178
 ventricular, 194
- Fibrosis of arteries, *see* Arterio-
 sclerosis
 bundle, 120, 123
 heart, 39
 coronary disease and, 40,
 364
 distribution of, 39
 dystrophic, 45
 in syphilis, 53
 inflammatory, 47
 ischemic atrophy, 32
 mixed forms of, 50
 myomalacia cordis and, 42
 para-arterial, 44
 peri-arterial, 46

- Fibrosis of heart, trauma and, 52
tuberculosis and, 52
tumours and, 52
- Field of response, 276
- Fingers, clubbing of, 245, 320, 462
- First rib sign, 424
- Flint's murmur, 338
- Flutter, auricular, 156
age incidence of, 157
arterial pulse in, 161, 164
auricular rate in, 156
circus movement in, 159
clinical features of, 159
diagnosis of, 169
digitalis in, 171, 382
etiology of, 157
experimentally, 158
heart-block and, 168
in diphtheria, 158
in exophthalmic goitre, 158
infarcts in, 163
morbid anatomy of, 158
prognosis of, 171
pulse rate in, 161, 164
quinidine and, 159, 197
symptoms of, 159
syncope in, 160
treatment of, 171
vagal compression in, 159, 169
ventricular contractions in, 160
- Focal lesions of arteries, 482
etiology of, 490
- Foci, septic, re-infection from, 224, 250
- Foramen ovale, 454, 458
- Foramina Thebesii, 41
- Fragmentation of cardiac muscle, 55
- Friction in pericarditis, 422
pleuro-pericardial, 423
- Functional mitral incompetence, 153, 291, 316, 318
murmurs, 153, 316
- Fundus oculi in angio-sclerosis, 514
- Gallop rhythm, 319
- Gall stones in heart disease, 289
- Galvanometer, string, 66
- Ganglion cells in sinus node, 15
- Gastro-intestinal symptoms of chronic valvular disease, 288, 304, 376
- Gerhardt's sign, 466
- Giddiness, arterio-sclerosis and, 512
extrasystoles and, 93
in "D.A.H.," 400
valvular disease, chronic, 290, 306
- Goitre, *see* Exophthalmic goitre
- Gonococcal endocarditis, 228
- Graham Steell's murmur, 154, 326
- Granular degeneration of myocardium, 33
- Graves's Disease, *see* Exophthalmic goitre
- Gumma of bundle, 123
of heart, 53
- Guy's pill, 378, 385, 537
- h* wave, 65, 82, 116
- Hæmaturia, 291
- Hæmopericardium, 409
- Hæmoptysis in aneurysm of aorta, 566, 576
auricular fibrillation, 193
flutter, 163
mitral disease, 283, 301
pulmonary infarct, 283, 301
valvular disease, chronic, 283
- Hæmorrhages in arterio-sclerosis, 513, 525
in myocardium, 51
- Headache, 290, 305, 512
- Heart-block, *see* Block, auriculo-ventricular, 118, 121
- Hemiplegia in aortic disease, 305
mitral disease, 293, 305
- Heterotopic beats, *see* Extrasystoles
- His, bundle of, *see* Bundle
- Histamine, action of, 528
- Hyaline degeneration of myocardium, 34
- Hydrogen ion concentration, 27
- Hyper-excitability, sympathetic, 78
- Hypermyotrophy, 475
- Hyperpiesis, 481
- Hyperthyroidism, tachycardia in, 79
- Hypertrophy of left ventricle, 275, 297
apex-impulse in, 309
electrocardiogram of, 70, 73
radiogram of, 328
sounds of heart in, 313
of right ventricle, electrocardiogram of, 71
signs of, 329
- Hypnotics in cardiac failure, 376
- Idio-ventricular rhythm, 114
- Infarct of heart, 42, 363, 366
in auricular fibrillation, 193
in aortic disease, 301
in mitral disease, 301
pulmonary, 301
- Infective diseases, acute, arterial disease and, 481
"D.A.H." after, 404
dilatation of heart in, 153

- Infective diseases, acute, endocarditis in, 223
 myocarditis in, 366
 pericarditis in, 411
 tachycardia in, 79
 tonicity depressed by, 153
- Inflow, venous, 25
- Influenza, 224, 406
- Inhibition, vagal, 21, 81, 115, 131, 201
- Insomnia, 290
- Intermission of pulse, 87, 118
- Intermittent claudication and arterio-sclerosis, 512
- Interpolated extrasystoles, 98
- Intestine, infarction of the, 286
- Intraventricular block, 138
- Iodides in aneurysm of the aorta, 583
 angina pectoris, 399
 arterio-sclerosis, 543
 cardiac failure, 378
- Irregularity of heart, extrasystolic, 87
 juvenile, 84
 perpetual, *see* Fibrillation, auricular
 respiratory, 83
 sinus, 83
- Irritable heart, *see* "D.A.H."
- Jaundice, bradycardia in, 82
 in heart-failure, 289
 in valvular disease, chronic, 289, 349
 pulse rate in, 82
- Jugular pulse in auricular fibrillation, 186
 extrasystoles, 96
 flutter, 164
 nodal rhythm, 211
 normal heart, 59
 sinus tachycardia, 80
- Jugulo-carotid curve, 59
- Juvenile irregularity, 84
- Kidney, functional efficiency of, 290
 in aortic disease, 267
 arterio-sclerosis, 477, 481
 mitral disease, 262
- Kussmaul's sign, 434
- Laryngeal, recurrent, nerve, *see* Recurrent laryngeal
- Larynx, paralysis of, 567
- Laxatives in arterio-sclerosis, 530
- Lead and arterio-sclerosis, 481
- Leucocytosis in acute endocarditis, 245
- Liver, cirrhosis of, 288, 353
 enlargement of, 288
 in dilatation of heart, 288
- Liver, enlargement of, in mitral disease, 302
 in tricuspid disease, 289, 310
 pulsation of, 289, 310
 tenderness of, 288
- Lungs, congestion of, 280, 300
 in aortic disease, 301
 mitral disease, 300
 infarcts of, 280, 301
 cedema of, 280
- Lymphadenoma, simulating aneurysm, 579
- Massage in acute endocarditis, 248
 in arterio-sclerosis, 531
 in chronic cardiac failure, 371
- Mechanical control of the heart, 24
- Medial calcification, 489
- Mediastinal tumour simulating aneurysm, 579
- Mediastinitis, chronic, 428
- Mediastino-pericarditis, 428
- Medulla, disease of the, and Adams-Stokes syndrome, 131
- Mental symptoms, 290
- Mercury in aortitis, 582
 in aneurysm of aorta, 583
 in arterio-sclerosis, 543
 in cardiac failure, 375, 376, 378
- Mesaortitis, 486, 546
- Mesarteritis, patchy, 485
- Metabolism, protein, and arterio-sclerosis, 528
- Miliary aneurysms in arterio-sclerosis, 495, 515
- Milk diet in arterio-sclerosis, 530
 in chronic cardiac failure, 372
- Milk spots, pericardial, 410
- Minute volume, 24
- Mitral incompetence, 329
 congenital, 467
 diagnosis of, 329
 etiology of, 257, 268
 functional, 153, 291, 316, 318
 murmurs of, 329
 prognosis in, 348
 signs of, 319
 ventricle, left, in, 293
- Mitral stenosis, auricular fibrillation and, 176, 326
 diagnosis of, 320
 electrocardiogram of, 70
 in congenital heart disease, 467
 murmurs of, 321
 prognosis in, 348
 radiogram of, 322
 signs of, 320

- Mitral valvular disease, acute endocarditis and,** 258, 270
auricular fibrillation in, 352
 blood-pressure in, 542
 cardiac rhythm in, 351
 causes of, 257, 268
 causes of symptoms, 275
 cerebral symptoms in, 289, 305
 chorea and, 273
 cardio-vascular degeneration and, 271
 dyspnoea in, 300
 embolism in, 304
 gastro-intestinal symptoms in, 288, 304, 376
 hæmoptysis in, 283, 301
 incidence of, 269
 mechanical strain and, 261
 morbid anatomy of, 252
 murmurs in, 321, 329
 oedema in, 287, 303
 onset of symptoms in, 293
 pain in, 302
 palpitation in, 284, 302
 prognosis in, 348
 renal disease and, 262
 rheumatism and, 270
 sex incidence of, 269
 symptoms of, 291
 syphilis and, 270
- Morphia in angina pectoris,** 397
 in cardiac failure, 375, 376
- Murmurs, cardio-pulmonary,** 318
 causes of, 314, 316
 conduction of, 313, 346
 diastolic aortic, 295, 337
 mitral, 322
 Flint's, 338
 functional, 154, 316, 341
 Graham Steell's, 154, 326
 in anæmia, 154, 316
 aortic incompetence, 295, 337
 stenosis, 317, 334
 auricular fibrillation, 186, 326
 congenital heart disease, 467
 depression of tonicity, 154
 dilatation of heart, 154, 316
 endocarditis, acute, 233, 241
 flutter, 161, 164
 mitral incompetence, 329
 stenosis, 321
 patent ductus arteriosus, 464
- Murmurs, in patent foramen ovale,** 458
 pulmonary incompetence, 343
 stenosis, 342, 462
 septal defects, 458, 460
 tricuspid incompetence, 344
 stenosis, 345
 musical, 318, 337
 presystolic, 292, 321, 338, 345
 systolic, 296, 315, 316
- Muscle of heart, conductivity of,** 113
 contractility of, 142
 excitability of, 87
 diseases of, 31
 layers of, 1
 properties of, 19
 tone of, 151
- Myocardial failure,** 358
 signs of, 367
 treatment of, 370
- Myocarditis, acute,** 222, 366
 alternation in, 146
 auricular fibrillation in, 176
 chronic, 358
 symptoms of, 358
 treatment of, 370
 flutter in, 158
 in endocarditis, acute, 222
 nodal rhythm in, 200
 subacute, 48, 158, 176, 244, 278, 355
- Myocardium, atrophy of,** 32
 cloudy swelling of, 33
 coronary arteries and, 361
 dilatation of, 151, 152
 diseases of, 31
 failure of, 358
 fatty degeneration of, 35, 359
 infiltration of, 34, 360
 fibroses of, 39, 364
 granular degeneration of, 33
 hyaline degeneration of, 34
 hypertrophy of, 31
 infarction of, 42, 366
 infective diseases and, 49, 358
 inflammation of, *see* Myocarditis
 ischæmic atrophy of, 32
 layers of, 1
 para-arterial fibrosis of, 44
 peri-arterial fibrosis of, 46
 rheumatic nodules in, 48
 rheumatism and, 48
 syphilis of, 53
- Nativelle's digitalin,** 195, 385
- Negativity, site of primary,** 20
- Nephritis, acute, blood-pressure in,** 533
 chronic, alternation in, 147
 aortic disease in, 267

- Nephritis, chronic, arterio-sclerosis and, 476
 blood-pressure and, 476
 mitral disease and, 264
 pericarditis in, 414
 in subacute endocarditis, 267
- Nerve fibres in bundle, 16
 in nodes, 15, 16
 optic, 514
 recurrent laryngeal in aneurysm of aorta, 567
 mediastinal tumours, 569
 mitral stenosis, 284
 sympathetic, *see* Sympathetic
 vagus, *see* Vagus
- Nervous control of heart, 21
- Neurasthenia, "D.A.H." and, 403
 pulse in, 78
- Neurogenic theory, 18
- Nitrite, amyl, 397
- Nitrites, uses of, 399, 536
- Nodal bradycardia, 205
 extrasystoles, 105
 rhythm, 198
 asphyxia causing, 199
 atropine and, 201, 205
 auriculo-ventricular bundle in, 201
 auriculo-ventricular node in, 198, 206
 cardiac weakness in, 213
 clinical features of, 201
 etiology of, 198
 experimentally, 199
 in endocarditis, 200, 205
 morbid anatomy of, 200
P-R interval in, 211
 rate of heart in, 201, 208
 vagal inhibition and, 199
- tachycardia, 205, 207
 continuous, 205
 paroxysmal, 207
- Node, auriculo-ventricular, lesions of, 122, 200
 position of, 7
 stimulus production in, 20, 198
 structure of, 15
 vagal action on, 22
 warming of, 199
- sinus, 6
 cooling of, 199
 excision of, 199
 function of, 77, 198
 ganglion cells in, 15
 inhibition of, 22, 81, 199
 nerve fibres in, 15
 site of, 6
 structure of, 15
 vagal action on, 22
- Nodules, rheumatic, 48
 subcutaneous, 234
- Obesity, heart in, 34, 360
- Ocular signs of aneurysm of aorta, 569
 arterial disease, 514
- Edema in aortic disease, 303
 auricular fibrillation, 179
 depression of tonicity, 153
 dilatation of heart, 153, 287
 flutter, 163
 mediastino-pericarditis, 436
 mitral disease, 303
 tricuspid disease, 341
 valvular disease, chronic, 287, 303
 pulmonary, 280
 renal, 303
 sodium chloride and, 373
 treatment of, 375
- Esophagus, compression of, by aneurysm, 570
 in pericarditis, 419
 hæmorrhage from, 570
- Oliver's sign, 567
- Ophthalmoscopic signs of arterial disease, 514
- Orthodiagram of heart in, aneurysm of aorta, 564
 aortic incompetence, 335
 aortic and mitral incompetence, 336
 auricular fibrillation, 185
- Orthopnoea, 280
- Ostium primum, 454, 457
 secundum, 454, 458
- Ouabine, 386
- Output of heart, systolic, 24
- Over-strain, cardiac, 153, 279
- Oxygen; carriers, 30
 in treatment, 375
 saturation of blood, 29
- P* deflexion, 68
- Pain, anginal, 388
 epigastric, 286, 302, 303
 in aneurysm of aorta, 552, 572, 573
 aortic valvular disease, 302
 aortitis, 578
 "D.A.H.," 400
 extrasystoles, 93
 mitral disease, 302
 pericarditis, 418
 valvular disease, 286
 referred, 395
- Palpitation, extrasystoles causing, 93
 in auricular fibrillation, 178
 "D.A.H.," 400
 flutter, 161

- Palpitation in valvular disease, 284, 302
- Paracentesis pericardii, 450
- Paradoxical pulse, 149, 434, 465
- Paroxysmal tachycardia, 198, 207
- auricular fibrillation and, 177
 - auricular flutter and, 170
 - cardiac reserve in, 208
 - cervical curve in, 211
 - etiology of, 198
 - nodal rhythm in, 207
 - prognosis in, 212
 - pulse-rate in, 211
 - stimulus production in, 198
 - symptoms of, 207, 208
 - treatment of, 213
- Patent ductus arteriosus, *see* Ductus arteriosus
- Patent foramen ovale, 458
- Pause, post-extrasystolic, 88, 96, 105
- post-paroxysmal, 100, 211
- Percussion of heart, 312
- Pericarditis, acute, 408
- acute rheumatism and, 411
 - chorea and, 413
 - diagnosis of, 425
 - effusion in, 420, 421
 - etiology of, 411
 - fibrinous, 422
 - hæmorrhagic, 409, 421
 - incidence of, 415
 - infections and, 411, 414
 - "milk-spots" and, 410
 - morbid anatomy, 408
 - onset of, 418
 - paracentesis in, 450
 - prognosis in, 427
 - pulse in, 419, 425
 - purulent, 421
 - pyæmia and, 414
 - renal disease and, 414
 - rheumatism and, 411
 - serous, 409
 - signs of, 422
 - symptoms of, 417
 - terminal, 414
 - treatment of, 449
 - tuberculous, 410
 - X-ray examination in, 425
- Pericardium, adherent, 428
- Broadbent's sign in, 433
 - cardiolysis in, 451
 - diagnosis of, 431
 - etiology of, 429
 - indurative mediastino-pericarditis, 428
 - mediastino-pericarditis, 428
 - morbid anatomy, 428
 - Pick's disease, 430, 435
- Pericardium, adherent, pulse in, 434
- signs of, 431
 - symptoms of, 430
 - treatment of, 451
 - tuberculous, 429
- Petechiæ in endocarditis, 245
- Petit mal in valvular disease, 306
- Phono-cardiograms, 75, 462
- Phrenic nerve, paralysis of, 570
- Physical signs in aneurysm of aorta, 551
- aortic incompetence, 336
 - aortic stenosis, 333
 - mediastino-pericarditis, 431
 - mitral incompetence, 329
 - mitral stenosis, 320
 - myocardial failure, 367
 - pericarditis, 422
 - pneumo-pericardium, 443
 - pulmonary incompetence, 343
 - pulmonary stenosis, 342
 - tricuspid incompetence, 343
 - tricuspid stenosis, 345
- Pick's disease, 430, 435
- Plasma, blood-, alkali reserve of, 27
- Plateau, systolic, 58
- Pleuro-pericardial friction, 423
- Pneumonia and endocarditis, acute, 224
- pericarditis, 414, 416, 420
- Pneumo-pericardium, 442
- Poisoning, tachycardia in, 79
- Polycythæmia, blood-pressure in, 479
- in cyanosis, 30
 - pulmonary stenosis, 462
- Polygraphic curves, interpretation of, 56
- Polyorrhomenitis, 430
- Polyserositis, 430
- Post-extrasystolic beat, 88
- pause, 88
- Post-paroxysmal pause, 100, 211
- Posture, pulse-rate and, 77
- Potassium iodide, *see* Iodides
- P-R interval, in auriculo-ventricular block, 115
- nodal rhythm, 203
 - normal, 75
 - prolongation of, 115
- Preponderance, ventricular, 70
- Presphygmic interval, 58, 115
- Pressure, blood-, *see* Blood-pressure
- intracardiac, and extrasystoles, 90
 - intracranial, bradycardia and, 82
- Presystolic murmur, 292, 321, 345
- disappearance of, 186, 326
- Primitive aorta, 453
- ventral aortæ, 453

- Primitive heart, structure of, 5, 453
 tissue of heart, 5, 453
- Prognosis of chronic valvular disease, 348
 age and, 355
 cause of failure and, 354
 degree of compensation and, 353
 disease of other viscera and, 353
 jaundice and, 349
 nature of lesion and, 349
 rhythm of heart and, 351
 sex and, 356
 site of lesion and, 350
 social conditions and, 357
- Prognosis in aneurysm of aorta, 581
 aortic incompetence, 350
 auricular fibrillation, 192
 congenital disease of heart, 349, 468
 endocarditis, acute, 246
 extrasystoles, 110
 flutter, 171
 heart-block, 132
 irregularity, sinus, 86
 nodal rhythm, 212
 paroxysmal tachycardia, 212
 pericarditis, 427
 pulsus alternans, 150
 valvular disease, chronic, 348
- Protein metabolism and arterio-sclerosis, 528
- Pulmonary artery, development of, 454
 atresia, 461
 incompetence, 341, 343
 congenital, 462
 pulsation of, 311
 stenosis, 342, 346
 congenital, 460
 clubbing of fingers in, 462
 etiology of, 455, 460
 murmur in, 462
 polycythæmia in, 462
 signs of, 462
- Pulsation, arterial, 59
 epigastric, 310
 of aneurysm of aorta, 554, 563
 aorta, abdominal, 575
 aortic incompetence, 578
 conus arteriosus, 311, 466
 jugular veins, 186
 liver, 289, 310
 pulmonary artery, 311
 ventricle, right, 309, 310
 præcordial, 307
- Pulse, alternation of, 143
 anacrotic, 335
 bigeminal, 97
- Pulse, collapsing, 338
 Corrigan's, 338
 deficit, 184
 dirotic, 368
 extrasystolic, 93
 in aneurysm of aorta, 558
 aortic incompetence, 338
 aortic stenosis, 335
 auricular fibrillation, 182
 Cheyne-Stokes breathing, 149, 281, 282
 endocarditis, acute, 230
 fever, 79
 flutter, 161, 164, 168
 heart-block, 119, 124
 mediastino-pericarditis, 434
 mitral disease, 327, 330
 paroxysmal tachycardia, 208
 pericarditis, 419
 Adams-Stokes syndrome, 130
 irregularity of, 83, 174
 palpation of, 499
 paradoxical, 149, 434, 465
 pressure, 297, 338, 340
 rate of, variations in, 77, 83
 respiratory irregularity of, 84
 trigeminal, 97
- Pulsus alternans, 143
 bigeminus, 97
 paradoxus, 149, 434, 465
 in adherent pericardium, 434
- Pupils in aneurysm of aorta, 569
- Purgatives, 378, 530
- Purkinje fibres, 10
- Pyæmia, endocarditis in, 235
 pericarditis in, 414
- Pyo-pericardium, 444
- Q* deflexion, 69
- Q-R-S* group, 69
 in branch block, 133
 in intraventricular block, 139
 normal, 69, 70
- Quinidine in auricular fibrillation, 196
- R* deflexion, 69
- R-T* period, normal, 74
 prolongation of, 74
- Radiograms of aneurysm of aorta, 560, 561, 562, 563, 571
 aortic incompetence, 563
 auricular fibrillation, 185
 hypertrophy of left ventricle, 328
 mediastino-pericarditis, 441
 mitral stenosis, 322
 pericardial effusion, 426
 pneumo-pericardium, 447, 448

- Radioscopy of aneurysm of aorta, 559
 aortic incompetence, 564
 auricular fibrillation, 184
 auriculo-ventricular block, 127
 congenital disease of heart, 468
 flutter, 169
 heart, 559
 heart-block, 127
- Rate of heart in auricular fibrillation, 182, 194
 cardiac failure, 368
 flutter, 161, 164, 172
 heart-block, 124
 nodal rhythm, 201, 208
 sinus bradycardia, 81
 sinus tachycardia, 79
- Recurrent laryngeal paralysis in aneurysm of aorta, 567
 mediastinal tumours, 569
 mitral stenosis, 284
- Reduplication of first sound, 319
 second sound, 319, 327
- Referred pain, 395
- Reflex origin of extrasystoles, 91
- Refractory period, 17, 159, 177
- Regulation of heart, 21
 mechanical, 24
 nervous, 21
- Reiss's sign, 435
- Renal disease and aortic disease, 267
 arterio-sclerosis, 506
 high blood-pressure, 262, 476
 mitral disease, 262
 pericarditis, 414
- Reserve power of heart, 25, 353
- Residual blood, 25, 153
- Respiration in heart disease, 27, 29, 280
- Respiratory exchange, 29
 irregularity of pulse, 83
- Response, field of, 276
 to exertion, 78, 353
- Rest, value of, 370
- Retinal hæmorrhages, 517
 vessels, 514
- Retinitis, 517
- Retrocardiac space, 561
- Retrosternal space, 561
- Rheumatic fever, *see* Rheumatism
 nodules, 48
- Rheumatism and aortic valvular disease, 270
 auricular fibrillation, 176
 endocarditis, 223, 225
 flutter, 158
- Rheumatism and heart-block, 124, 232
 mitral valvular disease, 270
 nodal rhythm, 200
 pericarditis, 411
- Rhythm, auriculo-ventricular, 198
 canter, 319
 coupled, 106
 gallop, 319
 nodal, 198
 normal, 20, 77
 sinus, 77
- Riva-Roccis phrygmanometer, 498, 521
- Roger, bruit de*, 460
- Rupture of aneurysm of aorta, 575
 heart, 54, 363
- S* deflexion, 69
- Salicylates in endocarditis, acute, 248
 pericarditis, acute, 450
- Salt free diet, 374
- Salvarsan, *see* Arsenic
- Scarlet fever and endocarditis, acute, 224
 pericarditis, acute, 414
- Semilunar valves, closure of, 58, 63
 mechanism of, 295, 296
- Sepsis and arterio-sclerosis, 525
- Septic foci, re-infection from, 224, 250
- Septal defects, 456, 459
- Septum, inter-auricular, defects of, 456
 development of, 453
 inter-ventricular, defects of, 459
 development of, 454
 membranous, development of, 454
 patency of, 459
 heart-block and, 460
 relation to bundle, 10
- Serum therapy in endocarditis, 249, 251
- Sex incidence of aneurysm of aorta, 547
 angina pectoris, 391
 auricular fibrillation, 175
 valvular disease, chronic, 269, 273, 356
- Sign, Broadbent's, 433
 Duroziez's, 339
 Gerhardt's, 466
 Kussmaul's, 434
 Oliver's, 567
 Reiss's, 435
- Silver, colloidal, in endocarditis, 251
- Sino-auricular block, 140
 node, *see* Node, sinus
- Sinus arrhythmia, 83
 bradycardia, 81

- Sinus bradycardia, diagnosis of, 83
 electrocardiogram of, 83
 etiology of, 81
 jugular tracing in, 83
 pulse in, 82
 third heart sound in, 82
 vagus nerve and, 83
 irregularity, 83
 age incidence of, 84
 atropine, action of, in, 86
 breathing and, 84
 electrocardiogram in, 85
 digitalis in, 86
 neurasthenia and, 84
 significance of, 85, 86
 node, *see* Node, sinus
 rhythm, 20, 77
 tachycardia, 78
 adrenalin causing, 78
 "D.A.H." and, 78
 emotion and, 78
 exercise and, 78
 in endocrine disturbance, 79
 exophthalmic goitre, 79
 sympathetic hyper-
 excitability, 79
 pulse-rate in, 79
 vagal paralysis and, 78
 Sinus venosus, 5, 453
 Skiagram, *see* Radiogram
 Sleep, promotion of, 375
 Sodium chloride, restriction of, 373
 Sounds, arterial, in aortic incom-
 petence, 339
 in extrasystoles, 94
 cardiac, 313
 characters of, 313
 conduction of, 313, 346
 duration of, 75, 76
 in auricular fibrillation, 186
 extrasystoles, 94
 flutter, 161
 heart-block, 118, 125
 phono-cardiogram of, 75
 reduplication of, 319
 time-relations of, 75
 Southey's tubes, 375
 Sphincter muscle, auriculo-ventricu-
 lar, 154, 291, 316
 Sphygmogram, *see* Pulse
 Sphygmomanometer, 498, 521
 Spinal cord in aneurysm of aorta, 571
 Sputum in heart disease, 283
 Squill, 385
 Staircase phenomenon, 123
 Stannius ligature, 113
 Starvation and arterio-sclerosis, 526
 Sternum, systolic retraction of, 433
 Stimulants, use of, 386
 Stimulus production, 77
 Stimulus production, depression of, 81
 in auriculo-ventricular node, 20,
 198
 bundle, 121, 122
 sinus node, 77
 normal, 77
 Stokes-Adams syndrome, *see* Adams-
 Stokes syndrome
 Strain, dilatation of heart and, 153,
 503
 valvular disease and, 261
 Stridor, 564, 580
 String galvanometer, 66
 Strophanthin, 172, 196, 385
 Strophanthone, 385
 Strophanthus, 385
 heart-block due to, 117
 in cardiac failure, 385
 in flutter, 171
 Strychnine in cardiac failure, 387
 Adams-Stokes syndrome,
 133
 Subaortic musculature, 296
 Subcutaneous nodules, 234
 Sudden death, 194, 350, 363, 384
 Sugar, blood-, in tachycardia, 79
 Supra-ventricular extrasystoles, 96
 Sympathetic, action on heart, 23
 extrasystoles and, 91
 hyper-excitability of, 78
 in aneurysm of aorta, 569
 nodal rhythm, 205
 over action, 24
 tachycardia and, 78
 Sympatheticotonia, 24
 Synchronous contraction of auricle
 and ventricle,
 coupled rhythms induced
 by, 107
 extrasystoles due to, 91
 in nodal extrasystoles, 105
 in nodal rhythm, 198
 Syncope, in Adams-Stokes syndrome,
 129
 flutter, 160
 sino-auricular block, 140
 valvular disease, chronic,
 306
 Syphilis, aneurysm of aorta and, 544
 aortic valvular disease and, 270
 aortitis and, 260, 485, 546
 arterial disease and, 485
 auricular fibrillation and, 176
 coronary arteries and, 278
 heart-block and, 122
 mitral disease and, 270
 of myocardium, 53
 treatment of cardiac, 386
 valvular disease and, 260, 270
 Systole, ventricular, duration of, 160

- Systolic blood-pressure, 338
 output, 24
 plateau, 58
- T* deflexion, 72
 abnormalities of, 72
 inversion of, 72
- Tabes dorsalis and aneurysm, 570
- Tachycardia, nodal, 199, 205, 207
 paroxysmal, 207
 sinus, 78
 ventricular, paroxysmal, 212
- Temperature in endocarditis, acute,
 234, 241
 pericarditis, acute, 420
- Theobromine, 386, 536
- Third heart sound, 65, 82, 319
- Thoracostomy, 451
- Thrill of, aortic stenosis, 334
 mitral incompetence, 329
 stenosis, 321
 pulmonary stenosis, 343, 462
 tricuspid stenosis, 345
- Thrombosis in auricles, 193
 in focal lesions of the arteries, 496
- Thyroid extract in arterio-sclerosis,
 543
 gland and tachycardia, 79
- Tobacco poisoning, angina pectoris
 and, 392
 bradycardia in, 82
 extrasystoles in, 91
 tachycardia in, 79
- Tone of heart muscle, *see also*
 Tonicity
 diastolic, 151
 of vagus, 21, 81, 83
 abolition of, 22
 increase of, 21
 waves, 151
- Tonicity of heart muscle, 151
 depression of, 153
 causes of, 153
 dilatation due to, 153
 digitalis and, 155
 murmurs in, 154
 signs of, 154
 symptoms of, 153
 treatment of, 155
- Tonsillitis and acute endocarditis, 224
- Tonus, *see* Tone and Tonicity
- Toxæmia, bradycardia in, 82
 extrasystoles in, 91
 tachycardia in, 79
 tonicity depressed by, 153
- Trachea, compression of, by
 aneurysm, 564
- Tracheal tugging, 567
- Tracing of apex impulse, 57, 62, 65
 arterial pulse, 57
- Tracing of cervical pulsations, 57,
 60, 62
 jugulo-carotid pulse, 57, 60,
 62
- Transmission time, 59
- Transposition of great vessels, 454
- Trauma and pericarditis, 414
- Treatment in adherent pericardium,
 451
 aneurysm of aorta, 582
 arterio-sclerosis, 521
 auricular fibrillation, 194
 cardiac failure, chronic, 370
 congenital heart disease, 468
 endocarditis, acute, 246
 extrasystoles, 112
 flutter, 171
 heart-block, 133
 paroxysmal tachycardia, 213
 pericarditis, 449
- Tricælian heart, 459
- Tricuspid incompetence, 341, 343, 351
 stenosis, 345, 467
- Trigeminal pulse, 97
- Trousseau's wine, 385, 537
- Truncus arteriosus, 454
 septum of, 454
- Tuberculosis, aortic aneurysm simu-
 lating, 579
 congenital heart disease and, 462
 of myocardium, 52
 pericarditis and, 410
 pulse in, 80
- Tumours of chest wall simulating
 aortic aneurysm, 577
 of heart, 52
- Typhoid fever, *see* Enteric fever
- Tyramine, action of, 528
- Uræmia, 510, 539
- Urea, blood-, 291
 concentration test, 290
- Urine in chronic valvular disease, 290
- v* wave, 61
- Vaccines in acute endocarditis, 249
- Vagotonia, 23, 81
- Vagus, action of, in auricular fibrilla-
 tion, 182, 192
 flutter, 166, 169
 heart-block, 115, 117, 133
 nodal rhythm, 211
 on heart, 21
 atropine, action of, on, 22
 compression of, 21, 118, 133, 192
 inhibition of, 21, 81, 115, 131, 201
 nodal rhythm and, 201, 211
 irregularity of heart and, 86
 stimulation of, causing brady-
 cardia, 81

- Vagus, stimulation of, causing depression of conductivity, 115, 117, 141
 extrasystoles, 91
 heart-block, 115, 117
 tone of, 21, 81, 83
- Valves, auriculo-ventricular, closure of, 61, 63
 blood-vessels of, 217
 re-infection of, 278, 355
 semilunar, closure of, 58, 63
 opening of, 58, 63
- Valvular disease, chronic, 252
 acute endocarditis and, 258
 congenital defects and, 268
 general cardio-vascular degeneration and, 271
 cardiac rhythm in, 279
 Cheyne-Stokes breathing, 281
 cough in, 283
 diagnosis, 307
 dyspnoea in, 280, 300
 etiology of, 257
 extrasystoles in, 90
 gastro-intestinal disturbances in, 288, 376
 mechanical strain and, 261
 morbid anatomy of, 252
 myocardial failure in, 277
 œdema in, 287
 pain in, 284, 286
 palpitation in, 284, 286, 302
 prognosis of, 348
 renal disease and, 262
 signs of, 307
 symptoms of, 275
 syphilis and, 260, 270
 treatment of, 370
- Varix, aneurysmal, 468
- Vaso-dilator drugs, 535
- Vaso-motor disturbances, 404
 extrasystoles due to, 91
 system, "D.A.H." and, 404
- Vasa vasorum, 471, 486
- Veins, jugular, *see* Jugular pulse
- Venæ cavæ, compression of, 554
- Venesection, 585
- Venous blood, 29
 inflow, 25
 pulse, *see* Jugular pulse
- Ventricles, dilatation of, *see* Dilatation of heart
 filling of, 25
 hypertrophy of, *see* Hypertrophy in aortic disease, 297
 output of, 24
- Ventricular deflexions, 69
 extrasystoles, 97
 preponderance, 70
 tachycardia, paroxysmal, 212
- Vertigo, *see* Giddiness
- Viscosity of blood, 478
- Volume of heart, diastolic, 151
- Wassermann reaction in aneurysm of
 aorta, 546
 aortic incompetence, 271, 386
 mitral disease, 261
- Water-hammer pulse, 338
- Waves, *a*, 61
c, 60
h, 65, 82, 116
v, 61
- Work of ventricles, 24, 275
- Wounds of heart, 52
- x* depression, 63
- X-ray of aneurysm of aorta, 558
 aortic incompetence, 335, 564
 auricular fibrillation, 184
 flutter, 169
- y* depression, 63

